THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XVII. JULY, 1936 No. 65

Original Papers

SPINAL SYMPTOMS WITH LYMPHADENOMA

By

I. M. ALLEN AND J. O. MERCER, WELLINGTON, NEW ZEALAND

I. Introduction 1
II. Pathogenesis of Spinal Symptoms 1
III. Case Reports 5
IV. Pathological Commentary 9
V. Clinical Commentary 10
VI. Discussion 12
VII. Summary and Conclusions 14
References 15

INTRODUCTION

IN the course of lymphadenoma involvement of the central nervous system appears to be uncommon. Detailed observation shows however that the incidence of neurological complications is probably higher than is suggested by casual inspection. Ginsburg found clinical evidence of involvement of the brain or spinal cord in ten of 86 cases of Hodgkin's disease. On the other hand, Forrest found that during 20 years at the London Hospital only four cases had shown evidence of involvement of the spinal cord. In one of these cases autopsy revealed syringomyelic cavitation.

In a review of 37 cases of Hodgkin's disease with involvement of the central nervous system Johnsson found the brain involved in eight and the spinal cord in 29 cases. Weil, summarizing 46 cases of involvement of the spinal cord in Hodgkin's disease, found dorsal segments affected in 80 per cent., cervical segments in 16 per cent., and lumbosacral segments in 4 per cent.

PATHOGENESIS OF SPINAL SYMPTOMS

The spinal symptoms and the changes in the spinal cord are produced in various ways.

1. In a small number of cases the vertebræ are invaded by lymphadenomatous tissue. Destruction of the bone may be followed by dislocation of the spine and direct mechanical compression of the spinal cord. In Lemierre and Augier's case lymphadenomatous tissue had invaded the cervical vertebræ, the spine was dislocated and the spinal cord was compressed. In McCallum's case of typical Hodgkin's disease there was an area of softening in the ribs. The patient died suddenly. At autopsy the axis was found destroyed by erosion, the vertebral column was dislocated and the spinal cord was almost completely severed. A similar accident may have caused death in Paullin's case. The lower cervical and upper dorsal nerves on the right side were involved and the seventh cervical vertebra appeared to be destroyed. The patient developed what appeared to be an acute cerebral crisis and died.

Direct extension of lymphadenomatous tissue into the bodies of the vertebræ is probably more common than invasion of other bones because of the presence of large groups of lymph glands close to the vertebral column in the thorax and abdomen. In two of 13 cases analysed by Parkes Weber bodies of vertebræ were eroded and the spinal cord was compressed. In a case described by Forrest of a male, age 18 years, X-ray examination revealed extensive destruction of the upper dorsal and lower cervical vertebræ. In many cases, however, direct invasion of bodies of vertebræ does not occur even when large masses of diseased glands are adherent to the vertebral column in the thorax and abdomen. Parkes Weber and Bode, and East and Lightwood have described cases of this type.

Delius considered it possible that, after the vertebræ had been infiltrated, lymphadenomatous tissue might proliferate between the bone and the dura mater, press forward the dura and thus compress the spinal cord ; but cases in which this sequence can be demonstrated or even assumed are rare. That of Carslaw and Young is the only one in the available literature in which it was demonstrated anatomically that infiltration of the vertebræ was associated with involvement of the membranes as a cause of symptoms of abnormality in the spinal cord.

2. The commonest finding is masses of abnormal tissue in the neighbourhood of the vertebral column, epidural deposits of lymphadenomatous tissue and no invasion of the bone itself. In Parkes Weber's original case a young man with Hodgkin's disease affecting the glands of the neck developed paraplegia late in the course of the disease. Autopsy revealed lymphadenomatous infiltration of the epidural fat. In a further case reported by Parkes Weber and Bode enlarged retroperitoneal glands, partly necrotic, were firmly adherent to the vertebral column. The bone was unaffected. There was ' diffuse lymphogranulomatous infiltration of the epidural fat on the dorsal aspect of the whole of the thoracic spinal cord, especially in the upper part.' Epidural deposits were present without direct invasion of the bone or dura mater in seven of Parkes Weber's 13 cases. Weil found invasion of the epidural space by lymphadenomatous tissue in 85 per cent. of 46 cases. In a

further 7 per cent. he found some disease of the retroperitoneal and intrathoracic lymph glands, paraplegia and scar tissue in the epidural space. He interpreted this scar tissue as an indication that there had existed previously lymphadenomatous tissue capable of producing spinal cord symptoms.

Parkes Weber described the pathology in his original case as lymphadenomatous thickening of the periosteum of the vertebræ and quoted six cases (one doubtful) from other authors. In discussing the case reported with Bode however he drew a distinction between infiltration of the epidural space alone and the association with it in some cases of involvement of the periosteum in front of the epidural fat.

The manner and route of extension of lymphadenomatous tissue to the epidural space demand consideration. Infiltration of the bodies of the vertebræ and extension later to the epidural fat is obviously unusual. Equally improbable is extension by other than a direct route, for in a large number of cases abnormal tissue in the epidural space is associated with frank disease of glands in the neighbourhood of the vertebral column. Some observers have described lymphadenomatous tissue in the mediastinal and retroperitoneal glands, in the intervertebral foramina with the nerve-roots and in the epidural space, and have concluded that the abnormal tissue extends by a direct route from the affected glands through the intervertebral foramina to the epidural space. East and Lightwood, Delius, and Schaeffer and Horowitz demonstrated epidural deposits within the spinal canal and invasion of the intervertebral foramina. In East and Lightwood's case the lymph glands were involved generally and the back muscles were infiltrated with lymphadenomatous tissue which formed masses about the sixth and seventh and the first and second thoracic spines. At these levels the intervertebral foramina were full of deposit. A similar condition was present in Parkes Weber's Case 14. These workers also demonstrated invasion of the nerve-roots in the intervertebral foramina ; and M. J. Cooper concluded from a study of his case and of the literature that the nerve-roots afforded a channel by which lymphadenomatous tissue might reach the subdural space, probably through the lymph spaces of the roots rather than by the substance of the nerve-fibres.

Thus symptoms of paraplegia may result from direct compression of the spinal cord by epidural deposits of lymphadenomatous tissue.

3. Extension of lymphadenomatous tissue from the epidural to the subdural space is less common, but has been demonstrated by Urechia and Goia, Johnston, During and Delius. M. J. Cooper regarded the spinal dura mater as relatively more resistant to extension than the septa of the nerve-roots, and from examination of his case concluded that penetration to the subdural space was probably by the latter route.

4. Invasion of nerve-roots in the intervertebral foramina has been demonstrated by East and Lightwood, Delius, and Schaeffer and Horowitz. Craver and Haagensen found seven cases of secondary herpes zoster in 329

cases of lymphosarcoma, Hodgkin's disease and leukæmia, and suggested that it was the result of invasion of nervous structures by the tumour masses. In four cases the herpes and the tissue mass were in the same general anatomical region.

5. M. J. Cooper concluded that spinal symptoms resulted not only from compression of the spinal cord by epidural collections of lymphadenomatous tissue, but also from compression or invasion of bloodvessels which accompany spinal roots. In Walthard's case the changes in the spinal cord were greater than would have been expected from direct pressure by an extradural tumour. They were thrombosis of bloodvessels, inflammatory and necrotic changes, and extensive softening in the spinal cord. It was suggested that the sequence of events was invasion of vessels in the tumour mass by lymphadenomatous tissue, congestion of vessels in the spinal cord and degeneration within the substance of the spinal cord.

6. It has been pointed out by E. L. Cooper that in the minority of cases there is no evidence of pressure on the spinal cord or spinal roots, or of obstruction of the circulation. In 8 per cent. of 46 cases analysed by Weil lymphadenomatous tissue had not invaded the spinal canal. In one case described by Forrest the spinal symptoms were due to syringomyelia. In the others there are found either long-tract degeneration, changes resembling those of acute myelitis or changes which are difficult to interpret or to relate directly to the lymphadenoma. Parkes Weber and Bode suggest however that if the epidural fat were examined as a routine, even when no nervous symptoms were recorded, lymphadenomatous infiltration of it would be found more often.

In Forrest's case the columns of Goll in the cervical cord and the central part of the dorsal columns in the remainder of the spinal cord were slightly degenerated. The ventrolateral and left lateral pyramidal tracts in the upper cervical region, both lateral pyramidal tracts in the sacral region and the cells of the anterior horns throughout the spinal cord were similarly affected. Allan and Blacklock's case showed spastic paraplegia with sensory symptoms. Shortly before death the left arm and face became weak and hemiplegia developed. At autopsy there was found acute meningitis with purulent infiltration over the base of the brain and the upper cervical cord. On histological examination no lymphadenomatous tissue was found in any part of the central nervous system. Robinson treated a patient suffering from Hodgkin's disease for 12 years with X-rays and radium. X-ray examination showed increased density of the body of the eighth dorsal and fourth lumbar vertebræ. Symptoms of transverse myelitis developed rapidly and corresponded to the level of the ninth dorsal segment. Critchley and Greenfield drew attention to the appearance of the characteristic clinical picture of subacute combined degeneration of the spinal cord in association with Hodgkin's disease and quoted McAlpine's case.

Discussing the occurrence of spinal symptoms in the course of leukæmia

and chloroma Critchley and Greenfield found cell masses in relation to the dura mater in four personal cases, and considered that the supposition that paraplegia was produced by pressure was not altogether supported by microscopical examination. Infiltration of the spinal meninges was recorded in 12 cases of chloroma and five cases of leukæmia, and in some had involved the anterior and posterior spinal roots and later the neighbouring muscles outside the spinal column by way of the intervertebral foramina. The secondary changes in the spinal cord were due to anæmia, hyperæmia with the possibility of acute myelitis, or compression as a partial factor. It was possible that the partial anoxæmia inseparable from severe anæmia might in many cases contribute to the effect produced by compression or thrombosis of the spinal arteries, but it was improbable that any degree of anæmia compatible with life could, of itself, so lower the nutrition of the nervous tissues as to lead to its disintegration.

CASE REPORTS

The two cases described in this paper fall into the indefinite group of cases in which unequivocal evidence of Hodgkin's disease was associated with spinal cord symptoms without the extension of lymphadenomatous tissue inside the spinal canal.

Case I.—A male, age 34 years, was admitted on January 13, 1934. One week before he felt pain in the lower part of the back which he thought was due to jarring it at work. Since then he had felt tired and had suffered from vomiting, diarrhœa, loss of appetite and slight frontal headache. For three days vision had been dim. He had not felt perfectly well for 18 months. One year before, glands were noticed to be enlarged. They had varied in size since and were large for three weeks before admission.

He was pale and his skin was cold and moist. Temperature, pulse rate and respiration rate were normal. The heart was normal. There was fullness of the right lower chest, movement of this part with respiration was diminished, and breath sounds were faint at the base of the right lung. The abdominal wall was retracted. The edge of the liver was felt 1½ inches below the right costal margin; and the spleen was felt 1 inch below the left costal margin. Examination of the central nervous system revealed no gross abnormality. The right submandibular, left and right axillary, and left and right inguinal glands were enlarged. The superficial cervical, upper and lower deep cervical and supraclavicular glands were slightly enlarged. The glands were firm, elastic, only slightly tender, discrete and mobile. Nothing abnormal was found in the spine.

X-ray examination of the lumbar spine showed no gross abnormality. The fæces contained occult blood. The urine was acid and contained a moderate amount of albumen. The deposit contained numerous hyaline casts, a few polymorph cells and numerous cylindroids. The blood Wassermann reaction was negative. The total non-protein nitrogen was 43 mgm. per 100 c.cm. of blood. The red blood count was 5,900,000; the hæmoglobin percentage 122; and the colour index 1. The white blood count was 18,000 and a differential count showed the following percentages: polymorphs, 9; lymphocytes, 11; mononuclears, 1; eosinophils, 78; and basophils, 1. The red blood cells were normal in the stained film.

During the first week in hospital he complained of weakness and numbness of the hands and legs. When examined on the ninth day the functions of the cranial nerves were normal. In the upper limbs, motor power was diminished, especially in the right arm and both hands; muscle tone was lowered; the biceps-jerks were absent and the supinator- and triceps-jerks were faint; and sensibility for pinprick was diminished over both hands to the wrists. In the lower limbs motor power was poor for all movements and muscle tone was below normal. The knee- and ankle-jerks were not elicited and both plantar reflexes were flexor. Sensation for pinprick was diminished over a short stocking area to above the ankles; and appreciation of changes in position of the left toes was impaired. The abdominal reflexes were not elicited.

On the eleventh day the white blood count was 11,000. There were 28 per cent. of polymorphs, 7 per cent. of lymphocytes, and 64 per cent. of eosinophils. The red blood cells showed slight changes in size and colour. On the thirteenth day cerebrospinal fluid was withdrawn from the fourth lumbar interspace. Jugular compression increased the pressure of the fluid. The fluid was slightly yellow and a small web of clot appeared in it on standing. The Pandy test showed a marked increase of globulin. There were six small round cells per c.mm. The Wassermann reaction was negative.

The patient was examined personally 14 days after admission. Four days before he was described as very weak, and the left upper eyelid drooped. On the day of examination he complained of sensations of pins and needles in the hands. The patient was in distress and examination was limited. The functions of the cranial nerves were normal. In the upper limbs motor power was much limited. The patient could only lift both hands and let them fall. Muscle tone was poor. The supinator- and biceps-jerks were not elicited and the triceps-jerks were faint. The Mayer thumb-reflex was present on both sides. Sensation for cottonwool, pinprick, deep pressure, vibration and changes of position was normal. In the lower limbs motor power was limited to the following movements. He could dorsiflex the feet slowly, plantar-flex them slowly and lift the knees about 1 inch from the bed. Other movements were impossible. Muscle tone was poor to flaccidity. The knee- and ankle-jerks were not elicited; and stimulation of the soles of the feet was followed by a faint movement of flexion. Sensation for cottonwool and pinprick was normal. Deep pressure was not appreciated. Vibration was not felt over the right malleoli and only faintly over the left malleoli. Attempts to recognize changes in position of the toes resulted constantly in gross errors. The abdominal reflexes were not elicited. The abdominal wall was held rigid and there was no movement with respiration below the costal margins. The patient was breathing with difficulty. Inspiration was forced and with each inspiratory movement the accessory muscles became tense. With each inspiration there was general expansion of the chest above the level of the third or fourth ribs, while below that level there was no evidence of action of the intercostal muscles or of the diaphragm.

During the first week in hospital a gland was removed from the right inguinal region for examination. The gland architecture was entirely altered. The differentiation between follicles and sinuses was lost. The gland showed a uniform structure consisting of lymphoid cells and many large clear cells, some of which had giant multilobed nuclei. The gland was infiltrated throughout with eosinophils and there was a moderate amount of eosinophilic infiltration of the capsule.

The temperature rose to 101° F. the day before death; the pulse rate rose steadily from 70 to 130 during the time in hospital; and from the seventh day the respiration rate rose to 25. The patient died 18 days after admission to hospital.

The body was examined nine hours after death. There were masses of discrete glands on both sides of the neck, in the axillæ and in the groins; and similar masses of soft and fleshy glands in the mediastinum along the aorta, at the hilum of the lungs and at the root of the mesentery. The spleen was enlarged and weighed 2 lb. 6 oz.

The liver was enlarged and soft, and showed pale nodules on the surface. The internal organs showed some degree of decomposition, the lungs were congested and there was nothing abnormal in the heart. The spinal column was normal. The brain was congested.

There were no naked-eye changes present in the spinal cord or its coverings. No lymphadenomatous deposits could be found in the neighbourhood of the intervertebral foramina. The dura was of normal thickness and appearance. The cord itself presented no areas of softening and the normal outline in cross-section was preserved throughout its length. The microscopic appearances showed little departure from the normal. There was congestion of the small vessels, both in the cord substance and in the pia-arachnoid. This was associated with some œdema in all regions. In the midthoracic region there were isolated lymphocytes in both the white and grey matter; but neither perivascular cuffing nor definite inflammatory collections were present. There was no tract degeneration. In the thoracic segments the anterior horn cells were spherical in shape but no nuclear changes could be demonstrated.

Summary of Case I.—In the course of an illness of short duration, during which sections of a lymph gland showed changes characteristic of Hodgkin's disease, there appeared symptoms of interruption of the spinal cord. No collections of lymphadenomatous tissue were found in the intervertebral foramina or within the spinal canal. The changes in the spinal cord were very slight compared with the gross effects observed clinically.

Case II.—A male, age 53 years, was admitted on February 10, 1933. He complained of pins and needles in the lower limbs and weakness of the legs of three weeks' duration. Previous illnesses were erysipelas and an operation for hydrocele. On examination, glands below the left angle of the jaw were enlarged; there was a systolic murmur in the aortic region and the lower pole of the right testicle was swollen. The remainder of the clinical examination revealed no abnormality.

Before the patient came under personal observation the following special examinations were made. The urine was normal. The blood Wassermann reaction was negative. The red blood count was 5,070,000; the percentage of hæmoglobin 100 and the colour index 1. The white blood count was 7,400 and a differential white count showed the following percentages: polymorphs, 62; lymphocytes, 24; eosinophils, 3; and basophils, 1. The red blood cells were normal. The pressure of the cerebrospinal fluid was increased by jugular compression. The fluid was clear and contained 17 small round cells per cubic millimetre. The Pandy test showed a marked increase of globulin. The Wassermann reaction was negative. Histological examination of a gland from the right groin showed the following. The gland was swollen and its structure was abnormal. Isolated germ follicles were absent, and diffuse endothelial hyperplasia was apparent throughout the tissue. A few large mononuclear cells closely resembling Dorothy Reed cells were seen.

On February 23, the glands of the axillæ and groins were enlarged. The lower limbs were becoming weak. On examination, the functions of the cranial nerves were normal. Sensation for pinprick was impaired over the hands and forearms. Motor power was diminished in the lower limbs. The right knee-jerk was more active than the left; and both plantar reflexes were extensor. Sensation for pinprick was diminished over the feet.

The patient was examined personally on March 3. Cooperation was good but failed steadily during the examination. The voice was weak and there was some weakness of the facial muscles for voluntary movements. Otherwise the functions of the cranial nerves were normal. In the upper limbs motor power was moderate and

muscle tone was diminished. The supinator- and biceps-jerks were not elicited, and the triceps-jerks were present. Sensation for cottonwool, pinprick, deep pressure, vibration and changes of position was normal. The abdominal reflexes were not elicited. In the lower limbs only slight and limited movements were possible at the knees, ankles and toe joints. Muscle tone was diminished to flaccidity. The knee- and ankle-jerks were present and equal; both plantar reflexes were extensor. Sensation for cottonwool and pinprick was normal; that for deep pressure, vibration and changes in position was absent. The submaxillary, upper cervical, epitrochlear and inguinal lymph glands were slightly enlarged. The abdomen was distended; no enlargement of the spleen was found. The heart and chest were normal. The bladder functions were undisturbed.

On March 14, the patient's general condition was unchanged. The voluntary movements of the lower limbs were less than before. There was no difficulty with the bladder. Two days before, spontaneous sensations of numbness and pins and needles had disappeared from the upper and lower limbs. A loose cough had appeared. On examination, the functions of the cranial nerves were unchanged. In the upper limbs motor power and muscle tone were decreased. Movements of the fingers, hands and forearms were weak and clumsy. The supinator- and biceps-jerks were not elicited; the triceps-jerks were present; and the Mayer thumb reflexes were absent. Changes in position of the fingers were not recognized. Other types of sensation were normal. In the lower limbs only faint movements were possible at the knee and ankle joints. The tone of the extensor muscles at the ankles was increased; elsewhere the muscles were flaccid. The knee-jerks were present; the ankle-jerks were not elicited; both plantar reflexes were extensor. Sensation for deep pressure, vibration and changes in position of the toes was absent. The skin was glossy and firm and pitted on pressure. In the chest, at the left base expansion was diminished, the percussion note was dull, and breath sounds were faint. The white blood count was 8,600 and the percentage of eosinophils had risen to 13.

On March 22, the patient stated that he often had sensations of feeling solid objects in the right and left hands. He felt that a solid object was present in his hand and on looking was surprised to find that there was nothing there.

On April 17, it was noted that for two weeks wasting of the upper limbs was becoming evident. It affected chiefly the small muscles of both hands and to a lesser extent the muscles of the forearms and upper arms. It was more obvious distally and in the right upper limb than in the left. The supinator-, biceps- and triceps-jerks were active. The bladder functions were normal. The lower limbs were unchanged.

In the meantime examination of the blood and cerebrospinal fluid was repeated. The red blood cell count was 5,160,000, the percentage of hæmoglobin 100 and the colour index 1. The white blood count was 8,000, and a differential white count showed the following percentages: polymorphs, 51; lymphocytes, 23; eosinophils, 6; transitional cells, 5; and basophils, 4. The red blood cells were normal. The cerebrospinal fluid contained 23 small round cells per c.mm., and the Pandy test showed a moderate increase of globulin.

The condition of the patient then continued unchanged for 10 months with the following exceptions. In June, 1933, there was precipitancy of micturition for one week followed by a week during which there was delay in beginning micturition. In August, 1933, the bowels sometimes moved involuntarily. The bladder was unaffected. In November, 1933, there occurred a respiratory infection. Histological examination of an inguinal lymph gland showed changes similar to those at the previous examination. Masses of large clear cells like hyperplastic endothelial cells extended throughout the gland, as did also masses of eosinophils. The amount of fibrous tissue was insignificant. The large clear cells seen before were now numerous.

An attack of erysipelas of the face occurred in December, 1933. In February, 1934,

the patient complained of aching in the suprapubic region. There had been precipitancy of micturition. Blood was passed by the rectum. The upper limbs were recovering and the patient could use his hands. Only slight movements were possible in the lower limbs. In March, 1934, the knee- and ankle-jerks were elicited and both plantar reflexes were flexor.

In August, 1934, motor power was fairly good in the upper limbs and control over their movements was satisfactory. Motor power was improving in the legs and muscle tone was increased in the extensor muscles. The knee-jerks were very active and both plantar reflexes were extensor. Sensation for pinprick over both lower limbs was delayed. The red blood count was 4,300,000, the percentage of hæmoglobin 80 and the colour index 1. The white blood count was 12,000; the percentage of polymorphs was 58, of lymphocytes 26, and of basophils 16. The red blood cells varied in size and shape. The cerebrospinal fluid contained five small round cells per c.mm., and the Pandy test showed no increase of globulin.

The patient died on May 4, 1935. Post-mortem examination 18 hours after death showed the following. The lymph glands all over the body were enlarged to the size of a walnut, softened and matted together. When they were cut, they were very soft and the cut surface was of a peculiar grey colour. In the groin the enlarged glands were necrotic. The spleen was enlarged to three times the normal size and weighed 2 lb. The cut surface was red, the pulp was soft and there was no fibrosis. The liver was normal in size and showed only toxic changes. The left kidney was enlarged. Its cortex was swollen and contained numerous pinpoint abscesses. There was much pus in the left renal pelvis and ureter. The lungs were œdematous, and toxic changes were apparent in the heart muscle. There were no changes in the brain.

Nothing abnormal was discovered in the gross appearances of the spinal cord, meninges or vertebral column. Microscopic examination showed no tract degeneration. There was no excess of leucocytes in any part of the cord and no marked vascular changes could be demonstrated. There was some œdema of the cord in all parts. No histological changes could be demonstrated in the anterior horn cells.

Summary of Case II.—Within three weeks of the onset of an illness with enlargement of lymph glands there appeared symptoms of gross damage to the spinal cord. Section of a gland showed changes characteristic of Hodgkin's disease. Though some improvement of the neurological condition occurred, paraplegia persisted until death 28 months later. No collections of lymphadenomatous tissue were found in the intervertebral foramina or within the spinal canal. Neither naked-eye nor gross microscopical changes could be demonstrated in the spinal cord.

PATHOLOGICAL COMMENTARY

In the cases described the pathological findings were both negative and positive. In neither case were there involvement of bones of the vertebral column, collections of abnormal tissue in the epidural space or compression of the spinal cord. Bloodvessels were not obstructed. Within the spinal canal there were no abnormalities other than those of the pia-arachnoid and spinal cord. The positive pathological features differed somewhat in the two cases. In Case I the structure of the abnormal lymph gland was characterized by lack of definition, infiltration of the gland and capsule with eosinophil cells and the presence of many large clear cells with giant multi-

lobed nuclei. Other organs showed changes usually associated with an acute toxic process in the body. Changes in the spinal cord were diffuse and widespread. They consisted in extensive œdema, especially at the periphery and round bloodvessels, engorgement of bloodvessels, infiltration of the tissue with small round cells and changes in the nerve-cells, especially of the anterior horns. In Case II the structure of the abnormal lymph gland was characterized by lack of definition, diffuse endothelial hyperplasia and the presence of a few large cells closely resembling Dorothy Reed cells. The meninges and the spinal cord showed little abnormal. In neither case was there demyelination or degeneration of long tracts.

In Case I the changes were those of a very acute and active process affecting not only lymph glands and the spinal cord but also the other organs of the body. In Case II they were those of a more chronic process with secondary effects in the kidneys and no obvious end-results of an acute process in the spinal cord. There was a definite contrast between the effects of an acute tissue-response in the lymph glands, spinal cord and other tissues in the one case, and those of a more chronic process in the lymph glands and spleen in the other case. The most remarkable feature was the disparity between the severe clinical effects in both cases and the slight pathological changes in the spinal cord in Case I and the even slighter changes in Case II.

CLINICAL COMMENTARY

The first case was that of a man age 34 years who had not felt well for 18 months and in whom the size of lymph glands had varied for 12 months. Acute symptoms had been present for one week. The total non-protein nitrogen content of the blood was high and changes in the urine indicated functional damage to the kidneys. In the blood 78 per cent. of a white cell count of 18,000 were eosinophil cells—figures which fell 11 days later to 64 per cent. of 12,000. There appeared symptoms and physical signs of damage to nervous tissues ; at first spontaneous sensations at the periphery of the upper limbs with diminution or loss of tendon reflexes, diminution of motor power and muscle tone and loss of sensation for pinprick over glove and stocking areas in both upper and lower limbs ; and later loss of motor power below the upper dorsal segmental level, diminution of motor power, muscle tone and tendon reflexes in the upper limbs, and loss of sensation for deep pressure, vibration and changes of position in the lower limbs. The cerebrospinal fluid was slightly yellow, clotted on standing, and contained an excess of globulin and six small round cells per c.mm. The patient died 25 days after the onset of acute symptoms.

The histological changes in a lymph gland and the clinical findings were those of Hodgkin's disease. The neurological picture was that of an acute lesion of the lower cervical and upper dorsal segments of the spinal cord with an onset which suggested primary involvement of more peripheral nervous

structures. The changes in the cerebrospinal fluid suggested that the condition was probably inflammatory. The rapid enlargement of lymph glands for three weeks, the rapid lowering of the patient's resistance, the response of the blood with a white cell count of 18,000 and 78 per cent. of eosinophils, findings indicating acute lowering of renal function, the appearance of an acute lesion of the spinal cord with an increase of the globulin and cells of the cerebrospinal fluid, and intense infiltration of a lymph gland and its capsule with eosinophil cells—all pointed to the rapid activation in the body of a pathogenic agent which had already been attacking it in a mild way during the previous 18 months. The spinal cord symptoms would appear to have been the result of direct attack upon the spinal cord as well as upon tissues elsewhere in which the more characteristic response of the disease under consideration was evident.

In Case II the development of the condition and the clinical course were more prolonged. A man of 53 had spontaneous sensations in the lower limbs for three weeks. Submandibular lymph glands were enlarged. Five weeks from the onset there were changes in the lower limbs associated with damage to motor paths in the spinal cord and sensation for pinprick was diminished over the feet. Seven weeks from the onset, submandibular, axillary and inguinal lymph glands were enlarged ; motor power and muscle tone were diminished and tendon reflexes were diminished or absent in the upper limbs ; and in the lower limbs motor power and muscle tone were diminished, the plantar reflexes were extensor, and sensation for deep pressure, vibration and changes of position was absent. The blood contained 3 per cent. of eosinophils, increasing later to 13 per cent., and the cerebrospinal fluid contained excess globulin and 17 small round cells per c.mm.

In Case II the clinical picture was established in six weeks, whereas in Case I it was completely established within one week from the onset of the acute phase. In the former the changes in the blood were less pronounced, those in the glands were of a more chronic type and the changes in the spinal cord developed more slowly. During the first year physical signs in the upper limbs improved considerably, those in the lower limbs improved slightly, and symptoms of interference with bladder-controlling paths appeared for short periods. During the first two months the percentage of eosinophil cells in the blood again fell to 6, that of basophil cells rose to 4, and changes were still present in the cerebrospinal fluid. At the end of 18 months there were no changes in the cerebrospinal fluid, the percentage of basophil cells in the blood had risen to 16, and the neurological condition was stationary.

The clinical picture and changes in the lymph glands were those of lymphadenoma. The neurological picture was that of a lesion affecting the lower cervical and upper dorsal segments of the spinal cord, and changes in the cerebrospinal fluid favoured a lesion of an inflammatory type. There was evidence of activity in this lesion during the first three or four months, and after that the clinical effects of the lesion gradually subsided with

occasional waves of activity. At the end of 18 months there was no evidence of an inflammatory reaction, and thereafter the clinical course was largely that of a permanent neurological disability with intercurrent infections.

In Case II the complete clinical picture was a response less acute, more prolonged, and developing and subsiding more gradually than in Case I. The neurological aspect developed with moderate activity and subsided slowly with occasional waves of slight renewal of activity. Widespread tissue reaction was less evident than in Case I. In Case I there had been increasing waves of activity of the disease during 18 months and the final acute phase with neurological symptoms developed on that background; whereas in Case II the condition began apparently on a background of normal health only three weeks before admission to hospital. If, as seems probable from the tissue and clinical reaction in Case I, a pathogenic agent had become suddenly active or the defences of the patient lowered, the modifications in the clinical course and tissue reactions in Case II would be due to either less activity on the part of the pathogenic agent or more enduring resistance on the part of the patient. In both cases the phase of the disease which brought the patient under attention was much more acute than is usual with the disease. In Case I the patient died early, though paralysis of respiratory movements probably hastened that event, while in Case II a position was reached in which the process was checked at least temporarily except in the lymph glands themselves.

DISCUSSION

Gordon and van Rooyen have shown that intracerebral inoculation of rabbits with emulsions of lymphadenomatous tissue is followed by characteristic nervous lesions in the animal. After an incubation period of three or four days the animal develops signs of ataxia, incoordination, muscular spasms, head retraction and generalized encephalomyelitis, and usually dies. Some animals which are affected in a lesser degree survive and pass into a chronic condition of which general emaciation and progressive atrophy of muscles are the chief features. The duration of the condition thus produced in the rabbit varies from three days to a month or more. According to Gordon, ' post-mortem examination of the rabbits that die shows generally a pronounced degree of marasmus but no naked-eye lesion of any kind, except occasionally for some slight congestion of the meninges. . . . In spite of the striking clinical picture, sections of the earlier rabbits showed, as in the case of rabbits that die after cerebral inoculation with dermovaccine, a scarcity or absence of infiltrative lesions; but with the more active lymphadenoma material that killed the rabbits in four days, definite lymphocytic meningitis was present and a proportion of the bloodvessels in the cortex and elsewhere were seen to be surrounded with a perivascular cuff of lymphocytes.' These effects cannot be produced by the intracerebral inoculation of lymph glands affected with lymphosarcoma, leukæmia, carcinoma, tuberculosis or syphilis.

These conclusions have been confirmed by Gordon, Ogilvie and van Rooyen, Gow, and van der Hoelden and Hulst. The nature of the pathogenic agent is, however, undetermined. Gordon suggested that its behaviour is analogous to that of certain viruses in man and animals, whereas van Rooyen concluded that the effects produced in the rabbit neither confirm the possibility of their being the result of a specific toxic activity nor exclude them from being due to the action of a filterable virus. Friedemann and Elkeles produced similar effects in rabbits by intrathecal injection of sterile bone-marrow emulsions; and subsequently Friedemann showed that they may also be produced by the inoculation of emulsions of normal splenic tissue, bone-marrow and normal leucocytes. Similar material from cases of acute leukæmia and pernicious anæmia yielded characteristic effects. Mackenzie and van Rooyen showed that the encephalitogenic agent in lymphadenomatous glands is not identical with the proteolytic enzyme extracted by Jochmann and Lockemann from leucocytes, splenic tissue and bone-marrow, nor with several proteolytic enzymes described by other workers, and found it still impossible to define the pathogenic agent in lymphadenomatous glands.

In general, 8 per cent. of the cases of lymphadenoma with spinal symptoms analysed by Weil and, in particular, the cases described by Forrest and the present writers show many features in common with the clinical and histological effects produced by inoculation of rabbits with emulsions of lymphadenomatous tissue.

Under experimental conditions the tissue reaction in the central nervous system develops in the neighbourhood of the original inoculation; and in the clinical cases it appears in that portion of the central nervous system adjacent to the most pronounced response in lymph glands lying along the vertebral column. This selective localization of lesions in the central nervous system would appear to depend upon some local factor in addition to the general response in the nervous tissues and the body as a whole suggested in Case I. Extension along nerve-roots and especially the lymph channels associated with them may be the local factor concerned. This has been suggested as the route of extension in those cases in which lymphadenomatous deposits have been found outside and inside the dura mater, and some evidence has been adduced in support. With local lymph stasis, local extension of a pathogenic agent from affected lymph glands and in addition rapid activation of the agent itself or rapid lowering of the resistance of the body, that portion of the nervous tissue associated with development of the local factor would be most vulnerable to attack.

Both in the experimental animals and in the clinical cases described there was a decided disparity between the severe clinical pictures produced and the paucity of findings on anatomical and histological examination. In the animals, naked-eye examination showed only slight congestion of the meninges; in the human subject only in the acute case did naked-eye examination reveal œdema of the substance of the spinal cord and congestion

of the meninges. In the clinical case there was in addition excess of globulin and cells in the cerebrospinal fluid. In the more acute animal cases definite lymphocytic meningitis was present and some bloodvessels in the cortex and elsewhere were surrounded with a cuff of lymphocytes. In the more acute clinical case similar infiltration of the tissues in the neighbourhood of bloodvessels was seen in the affected parts of the spinal cord. In the more chronic animal cases, the animal recovered except for marasmus and muscular wasting, and anatomical examination revealed a scarcity or absence of infiltrating lesions. In addition, some of them were found when recovery was complete to be immune to a second dose of the same material administered in the same way though effects were produced in a control animal. In the more chronic clinical case the activity of the neurological lesion gradually subsided and, after occasional waves of slight renewal of activity, disappeared and left a stationary neurological disability. In spite of the progress of the disease in the lymph glands and spleen, there appeared no further renewal of activity in the spinal cord lesion. Detailed examination of the affected parts of the spinal cord revealed no obvious changes.

The resemblance of the effects in the experimental animal to those in the human subject is at least striking. While the data available leave it undetermined whether the encephalitogenic agent in lymphadenomatous tissue is the essential pathogenic agent of lymphadenoma, the association in the human subject of similar neurological findings and effects with active lesions of lymphadenoma suggests the presence of a factor common to both and the identification of it with a pathogenic agent.

SUMMARY AND CONCLUSIONS

1. The available information indicating the frequency and distribution of neurological lesions with lymphadenoma is summarized.

2. The methods by which spinal symptoms are produced by lymphadenoma are reviewed; and consideration is given to involvement of the vertebral column and its effects, epidural infiltration with lymphadenomatous tissue, extension of this tissue inside the dura mater, involvement of nerve-roots, obstruction of bloodvessels and compression of the spinal cord.

3. In 8 per cent. of cases of lymphadenoma with spinal symptoms the latter appear without compression of the spinal cord or any of the factors indicated under (2).

4. The clinical and pathological features of two cases of lymphadenoma with spinal symptoms are described.

5. The clinical and pathological findings favour the presence in lymphadenoma of a pathogenic agent which may be responsible under suitable conditions for the appearance of lesions in the tissues of the central nervous system.

REFERENCES

1 Allan, G. A., and Blacklock, J. W. S., *Glasgow Med. Jour.*, 1925, **103**, 115.
2 Carslaw, J., and Young, J. S., *ibid.*, 1927, **108**, 193.
3 Cooper, E. L., *Med. Jour. Austral.*, 1935, **1**, 585.
4 Cooper, M. J., *Jour. Amer. Med. Assoc.*, 1934, **102**, 917.
5 Craver, L. F., and Haagensen, C. D., *Amer. Jour. Cancer*, 1932, **16**, 502.
6 Critchley, M., and Greenfield, J. G., *Brain*, 1930, **53**, 11.
7 Delius, L., *Deut. Zeits. f. Nervenheilk.*, 1932, **125**, 142.
8 Düring, M., *Deut. Arch. f. klin. Med.*, 1918, **127**, 76.
9 East, C. F. T., and Lightwood, R. C., *Lancet*, 1927, **2**, 807.
10 Forrest, D., *ibid.*, 1927, **2**, 809.
11 Friedemann, U., *Brit. Med. Jour.*, 1934, **1**, 517.
12 Friedemann, U., and Elkeles, A., *ibid.*, 1933, **2**, 1110.
13 Ginsburg, S., *Arch. Int. Med.*, 1927, **39**, 571.
14 Gordon, M. H., *Report of the Rose Research on Lymphadenoma*, 1932.
15 Id., *Brit. Med. Jour.*, 1933, **1**, 641.
16 Id., *Proc. Roy. Soc. Med.*, 1934, **27**, 1035.
17 Gow, A. E., *ibid.*, 1934, **27**, 1045.
18 Johnsson, V., *Hygeia*, 1931, **93**, 39.
19 Johnston, J. M., *Pennsylvania Med. Jour.*, 1931, **34**, 877 (quoted ref. 4).
20 Laurent, L. P. E., *Proc. Roy. Soc. Med.*, 1931, **25**, 42.
21 Lemierre, A., and Augier, P., *Ann. d'anat. path.*, 1931, **8**, 916.
22 McAlpine, D., *Trans. Med. Soc. Lond.*, 1922, **45**, 20 (quoted ref. 6).
23 McCallum, W. G., *Jour. Amer. Med. Assoc.*, 1931, **96**, 2150.
24 Mackenzie, I., and van Rooyen, C. E., *Brit. Med. Jour.*, 1935, **1**, 406.
25 Ogilvie, R. F., and van Rooyen, C. E., *Lancet*, 1933, **2**, 641.
26 Paullin, J. E., *Jour. Amer. Med. Assoc.*, 1931, **96**, 2150.
27 Robinson, G. A., *ibid.*, 1928, **91**, 1548.
28 Schaeffer, H., and Horowitz, A., *Presse méd.*, 1930, **38**, 403.
29 van Rooyen, C. E., *Brit. Med. Jour.*, 1933, **1**, 50; 1933, **1**, 644; 1933, **2**, 562; 1934, **1**, 519.
30 Walthard, K. M., *Zeits. f. d. g. Neurol. u. Psychiat.*, 1924, **97**, 1.
31 Weber, F. Parkes, *Quart. Jour. Med.*, 1923–24, **17**, 1.
32 Weber, F. Parkes, and Bode, O., *Lancet*, 1927, **2**, 806.
33 Weil, A., *Arch. Neurol. and Psychiat.*, 1931, **26**, 1009.

ON SOME CONSTITUTIONAL ASPECTS OF CHOREA AND ON ITS SEQUELÆ*

By

E. GUTTMANN, London

If one sees many cases of chorea and investigates them in detail, one cannot help thinking that constitution plays a part of some importance in its ætiology. In textbooks one finds remarks to the effect that the patients had already exhibited neurotic traits before the onset of the disease. Furthermore, among relatives cases of chorea and other mental and nervous diseases and abnormal personalities are observed with some frequency, as far as a general impression goes. Kehrer has proposed to name the abnormal types in choreic families 'choreopathic personalities.' In doing so he took as his starting point Huntington families, but he expressly extended his idea to families with Sydenham's chorea. The definition of the choreopathic personality is not exact as yet, on either physical or psychical side, nor do we know whether the postchoreic personality which will be discussed later on is identical with the choreopathic personality or even similar to it.

It may be mentioned that the schizoid personality has been conceived along similar lines, viz. as a premorbid personality, as postschizophrenic defect and as a constitutional anomaly in schizophrenic families. There is moreover some constitutional relationship between chorea and schizophrenia. Looking through the families of some choreic patients I found some frequency of catatonic psychoses among their relatives. B. Schulz has confirmed these observations in material which was relatively small but elaborated on strict genetic lines. He found that the proportion of schizophrenics in the families of choreic patients is nearly twice as high as in the average population. Krauss also found in the families of 24 cases of chorea eight certain and three doubtful cases of schizophrenia. This author was the first to investigate systematically the postchoreic personality, earlier writers having been interested only in the neurological sequelæ of this disease, and that to a limited degree. Looking through the textbooks of neurology with regard to the prognosis of chorea one generally finds it stated that only a small proportion of the patients die, and that death in these cases is due to endocarditis or heart failure, the prognosis of severe cases generally depending on the

* With the support of the Rockefeller Foundation. From the Maudsley Hospital, London.

efficiency of the heart. As to the prognosis of mild cases little is to be found. One has to conclude that they recover; Russel Brain, however, says in his textbook: 'Chorea as such leaves no serious sequels, though some mental instability may persist for a long time, and slight involuntary movements may be perpetuated as a habit.'

In the last few years several after-histories have been published which give evidence that in some cases permanent disturbances of motility are left behind.

The present writer published in 1927 18 cases, nine of which showed motor anomalies for as long as 18 years after leaving hospital. Straus following up 23 cases saw choreiform, myoclonic or tic-like movements in 10 of them. Krauss observed hyperkineses in 17 cases out of 23 many years after the acute illness. Ossipowa noted in 60 per cent. of her cases residual motor symptoms, two or three years after their discharge. Straus was able to show that in a fair percentage of tiqueurs one finds chorea in the history; the more one directs one's attention to this, the more one is obliged to confirm his observation. As mentioned above, Krauss was the first to investigate systematically the psychic state after chorea. He regards the psychic picture as being similar to that of the schizoid psychopath. He finds the patients 'irritable and sensitive, seclusive, taking everything very seriously, querulous and suspicious . . . they are not very impressionable and little capable of emotional adaptation to their surroundings.' Besides the residual motor symptoms (tics, tremor, fidgetiness) he finds neurasthenic features (headaches, vertigo) and psychasthenic ones (forgetfulness, anxiety, lack of endurance, slowness of psychic acts). 'The postchoreics are not very vital or agile; they are rather quiet and listless persons.'

FREQUENCY

Chorea appears to be rather frequent in this country. Thus among the 2,026 in-patients of the Maudsley Hospital discharged between the years 1932–34 13 cases of acute chorea were found, while among the 22,000 admissions to Kraepelin's Psychiatric Clinic in Munich which had very similar material only 18 cases of chorea were noted from 1907 to 1922. Among the admissions to the Children's Hospital in Basle between 1910 and 1930 Krauss found only 50 cases, and in Eppendorf, the best-known German neurological hospital, from 1924 to 1933 only 41 cases of chorea were admitted among 16,000 patients. In England, on the other hand, Langmead (*L.C.C. Report*, 1910) in 2,556 children found 0·7 per cent. with evidence of chorea. Hadfield (*L.C.C. Report*, 1911) in 2,249 boys found 0·3 per cent., in 3,050 girls nearly 1·0 per cent. The *Annual Report of the L.C.C.*, 1934, mentions 1,094 cases of chorea.

RELATION TO PSYCHOSES

The frequency of chorea is also striking in histories and family histories of in- and out-patients at the Maudsley Hospital. This offered a new access

to the problems mentioned above, viz. the rôle of constitution in the ætiology of chorea and its sequels. Instead of following up patients who had suffered from chorea previously I looked through the in-patient records for three years to collect cases where chorea had occurred in the history or in the family tree.* Probably the incidence of chorea in the family of the average patient would not have been noted as regularly as in cases of those who had chorea themselves. Excluding the latter, there were four persons in whose families chorea was mentioned. In two their mother, and in two others a sister had suffered from St. Vitus' dance. Of the four patients in question three were admitted because of schizophrenia, and one because of depression. The numbers are too small to draw conclusions, but they gain some importance if one compares them with the investigation of the individual histories. In 24 of the records during the period mentioned chorea was found in the history. The following table shows the diagnosis of the cases :

Diagnosis.	Number.	Number of Discharges.
Schizophrenia	7	300
Depression	5	540
Hysteria	4	120
Anxiety State	4	140
Moral Abnormality	3	
Neurasthenia	1	
	24	

The figures are the more impressive if one compares them with those in the second column. This refers to the number of cases which had been admitted for the first time under this diagnosis. Without basing exact statistical conclusions on these numbers one can say roughly that *in the history of schizophrenics chorea is nearly twice as frequently noted as in depressives*, and that the figures are still higher in hysteria and anxiety states, the two most important neuroses.

But psychiatric diagnoses are only labels which do not characterize the individual case perfectly. Therefore the cases ought to be reviewed singly. In this way one cannot expect results of statistical value, but perhaps one will learn whether and how chorea modifies the clinical picture. A perusal of the records and after-histories, with regard to diagnosis, allows of doubt in only one of the schizophrenics (Case 2).

> In this case the state of the circulation suggests a symptomatic psychosis of the paranoid schizophrenic type accompanying heart failure. There is no possibility of finding the cause of the latter. Perhaps it had been the final effect of a choreic endocarditis, for as early as 1916 the patient had been rejected from the army because

* Only first admissions were used for technical reasons, and all cases of organic deterioration (general paralysis, arteriosclerosis) were excluded, as the data for childhood are generally not so complete, and the reliability of memory would be doubtful.

of his heart. On the other hand the result of the Wassermann reaction points to a syphilitic disturbance.

The sequence chorea—vitium cordis—symptomatic psychosis of paranoic schizophrenic type is illustrated by a case like St. B.*

Two depressions among the five cases mentioned above had subsequently to be eliminated. In the one case (Case 3) the psychosis started during a septic puerperium. The symptoms were depression and agitation, but also paranoid ideas and clouding of consciousness. The patient died in the psychosis. Also in the second case (no. 23) immediately after childbirth a symptomatic psychosis with disturbances of consciousness, incoherence, resistiveness, restlessness and paranoid ideas started. There was a mitral stenosis with dilatation of the heart as causal factor. This patient recovered. A third case (no. 22) with a similar history is still in a mental hospital and apparently shows a schizophrenic defect. Thus only two depressive cases have been left in which the patients previously suffered from chorea ; one of these (Case 21) is described in the record as having hysterical features. She exhibited a depersonalization state, without the characteristic traits of an endogenous melancholia. The fifth case (no. 4) is also rather an atypical depression. Furthermore the history of chorea in this case is not very definite.

A review of the anxiety states indicates that in one case the state is due to or can reasonably be considered as a psychogenic reaction. In two others the physical state is of considerable importance. One started during pregnancy, the other coincided with the chorea, but the last case (no. 17) may have been an endogenous depression.† Without overrating the exact proportions it seems possible to conclude that, as far as the endogenous psychoses are concerned, chorea is far more frequent in the history of schizophrenics than of manic-depressives (approximately 8 : 2).

In the literature on this subject there is only one remark about chorea in the history of schizophrenic patients. Schilder mentions a patient who ' had chorea in her childhood, and it is known that not only patients with catatonic troubles but also their relatives are comparatively often affected with chorea or choreiform troubles.' If one is not inclined to think of the coincidence as being due to pure chance one must consider the relationship of the constitutional factors underlying both diseases. It may be, on the one hand, that the disturbances produced by chorea play a part in the

* Endocarditis may be the link between chorea and other neurological symptoms, e.g. a case of Jacksonian epilepsy after an embolism apparently originating from choreic endocarditis has been observed. Later on the patient developed also generalized attacks.

† Recently I have observed in addition to the series of cases upon which this paper is based, several schizophrenics with chorea in the history, and one patient with mild reactive depression and no schizoid features, who had had chorea in her childhood. She had a tic as a motor after-effect. A sister of this patient suffered from a schizophrenic psychosis.

pathogenesis of the psychosis—which seems unlikely considering the statistical relations—on the other hand that they have a pathoplastic effect on the subsequent psychosis. I have mentioned above Krauss's investigations in which the choreic personality is not exactly distinguished from the post-choreic personality. The descriptions of the previous personality found in the histories are remarkably similar to what Krauss describes. But the evidence they supply is not decisive, as the periods before and after chorea have not been discriminated in this material, nor were data collected for this particular purpose. We find descriptions like the following (the first figure in the brackets gives the age at the time of the chorea, the second one the age at the onset of the disease which brought the patient into this hospital).

Case 1 (7, 22) ' delicate, reserved, suspicious.'
,, 2 (11, 39) ' seclusive, suspicious, unsociable, day-dreaming.'
,, 6 (childhood, 22) ' reserved, quiet, solitary.'
,, 9 (9, 24) ' particular, reserved, sensitive.'
,, 10 (6, 32) ' sensitive, nervous, seclusive.'
,, 22 (school, 36) ' reserved, shy, sensitive.'

As one sees, the chorea commences early, so that one can hardly find out whether a change of personality occurred or not. There was in only one case a definite statement to the effect that the patient had always been a difficult child, sulky and obstinate; in two other cases in which chorea occurred relatively late one can infer from the history that the description of the previous personality refers to the prechoreic period (Cases 7, 21). In the last case it is not quite clear from the description whether a previous attack of chorea had occurred while the patient was at school. In only one case (no. 24) is there evidence that a change of personality became noticeable after the chorea. In the record we find this: ' before 13 normal, gets on fairly well with people, but quarrels with other children. Between 13 and 16 energetic and sociable, fond of parties, lots of friends. First attack of chorea at 14, recurrence at $16\frac{1}{2}$. Since then has gradually become more difficult and quarrelsome. Criticizes people and is afraid people may criticize her.'

A collection of notes about the previous personality of cases admitted to hospital with chorea during the same period shows some similarly abnormal characters:

' Impulsive, obstinate, demanding, easily hurt, good opinion of herself.'
' Peculiar, difficult to manage, violent temper.'
' Obstinate, quick temper, sulky and disobedient.'
' Excitable, sensitive, conscientious.'
' Excitable, put off her balance, could not stand jokes and teasing.'
' Shy, quiet, passive.'

The results of Rorschach tests performed on the latest admissions of chorea point also in the same direction.

These abnormal traits in the prechoreic personality would point still more to a constitutional disposition than to an effect of the disease on

the personality. But we must bear in mind that the last-mentioned material must be used with special caution, as the cases have been admitted to the Maudsley Hospital as a rule not because of the chorea but for psychic complications.

The possibility of a postchoreic personality-change cannot, it is true, be excluded by the collection of this scanty material found in the records. There is still a possibility that minute changes have not been mentioned or have not been related to chorea. This possibility is to be stressed, since one sequela of chorea, viz. the motor instability, emerges even from these records which have been written without particular regard to this point.

DISORDERS OF MOTILITY

In eight cases motor symptoms like those we have seen as sequels of chorea have been described as existing before the psychosis. The relatives describe them as ' tics,' ' nervous,' ' twitching,' they call the patients fidgety, jumpy. In other cases during the psychosis ' jerky movements ' or similar symptoms are noted without its being possible to demonstrate whether they had existed before or were only developed during the disease. In either case the interpretation offered above would still stand. In a previous paper the present writer has shown that the motor instability left behind by chorea may be elicited by various noxæ, that agents of varying kinds, such as fatigue, intoxication or infection may produce choreiform disturbances of motility in persons who had previously suffered from chorea. It was mentioned in that paper that endogenous psychoses occurring in such patients may thus acquire a peculiar colouring.

Although no special attention has been paid to motor behaviour in the records upon which this study is based, motility disturbances were noted in 10 out of 13 endogenous cases. Sometimes they were simply described as catatonic symptoms, sometimes as mannerisms or as agitated, disconnected, jerky movements. This material is not ample enough to cast new light on the motility psychoses; Lewis and Minski discussed these questions in a publication on chorea psychoses a short time ago. But one might refer briefly to those cases in our material which do not belong to the endogenous psychoses. Can one demonstrate in them permanent residues of chorea ?

In all the four cases diagnosed as hysteria in this series, there is found evidence of motor disturbance.

> (No. 11) ' Face and limbs twitch.'
>
> (No. 12) ' Movements of hands and legs. Hysterical gait.'
>
> (No. 13) ' Trembling movements . . . coarse jumpy tremor suggestive of hysterical chorea.'
>
> (No. 24) Chorea accompanied by screaming attacks. . . . ' Her choreiform movements would cease with the utmost abruptness and it was thought that her condition was partly choreiform and partly hysterical.'

In all these cases the hysterical symptoms are much influenced and, indeed, partly determined by the existence of the residual motor symptoms. The question remains whether the tendency to produce conversion symptoms is based upon psychic factors or if the actual bodily condition (i.e. the choreic sequelæ) is responsible for it. In the light of the writer's previous investigations there is justification to stress the importance of the physical factors, even though it cannot be fully assessed in these cases.

Among the anxiety states one patient (no. 20) had been admitted because of complaints of fidgety movements. In the hospital she showed 'numerous irregular hysterical movements.' Another patient is described as 'excited, restless and uncontrolled.' As these features did not fit in with the clinical picture the doctor in charge was inclined to think of a hypomanic component; looking back one may suppose that there was a motility disturbance of postchoreic type which constituted the atypical traits. A third patient (no. 18) who suffered predominantly from fear of an undesired pregnancy had had two attacks of functional disturbance of gait prior to this neurosis. In the last case (no. 17), probably an endogenous depression, there was, as mentioned above, nothing but a marked disturbance of concentration which could remind one of the distractibility of choreics.

The pathoplastic significance of the postchoreic motor instability with regard to another disturbance has been described in a paper by Creak and Guttmann. It could be demonstrated in a group of patients with tics following chorea that the motor defect is of importance not only for the tic itself but also for the psychic phenomena connected with it. I have made some similar observations since and hope to be able to show in the course of further observations how the disturbance of motility merges into the complicated structure of neuroses and psychoses.

Motor behaviour as a special aspect of the personality deserves to be estimated more highly than it has been up to now. How important it is may be illustrated by the fact that the main stages of an individual's development can be defined by their particular motility. Also psychological types may be characterized by their motor qualities. In two quite recent publications use has been made of motility for definition of types. Earl distinguished over-movers and under-movers in morons, doing performance tests. Creak found among children with reading disabilities a group which was different from the others in respect to motility.

If one appreciates the significance of motor anomalies for such clinical purposes one will find interest not only in their descriptive side but also in their cause, pathogenesis and the interplay between them and the total constitution.

SUMMARY

Comparing the histories of psychotics, one finds chorea more frequently in schizophrenics than in manic-depressives. Also the premorbid personality

and heredity (previously investigated) point to a relationship of chorea with the schizophrenic group.

Chorea often leaves behind an abnormal motility which may enter as an important feature into the personality. This can be demonstrated in neuroses as a pathoplastic factor.

I wish to express my thanks to Dr. Mapother for his kind permission to use the material of the Maudsley Hospital and to publish these cases.

REFERENCES

CREAK, M., 'Reading difficulties in children,' *Arch. of Dis. in Childhood*, 1936, **11**, 143.

CREAK, M., and GUTTMANN, E., 'Chorea, tic, compulsive utterances,' *Jour. Ment. Sci.*

GUTTMANN, E., 'Beobachtungen bei Chorea minor,' *Zeits. f. d. g. Neurol. u. Psychiat.*, 1927, **107**, 584.

KEHRER, F., *Erblichkeit und Nervenleiden. I. Chorea, Myoklonie, Athetose*, 1928.

LEWIS, A., and MINSKI, L., 'Chorea and Psychosis,' *Lancet*, 1935, 356.

OSSIPOWA, 'Ueber die konstitutionellen Eigenschaften bei Chorea minor,' *Zeits. f. d. g. Neurol. u. Psychiat.*, 1930, **125**, 69.

SCHILDER, P., *Brain and Personality.*

SCHULZ, B., 'Beiträge zur Genealogie der Chorea minor,' *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, **117**, 288.

STRAUS, E., 'Untersuchungen über die postchoreatischen Motilitätsstörungen, insbesondere die Beziehungen der Chorea minor zum Tic,' *Monats. f. Psychiat. u. Neurol.*, 1927, **66**, 261.

APPENDIX

Case Histories

Case St. B.—Admitted April 8, 1935. *Complaint:* Depression accompanied by ideas of persecution and poison. *Family history:* Very poor family. Periods of great poverty owing to unemployment. Mother and two siblings said to suffer from nerves. No history of actual mental breakdowns. *Personal history:* Brought up in great poverty; night terrors and bed-wetting when young; bullied at school, reached top standard. Started work at 13. At age of 15 employed by booksellers and remained with firm for 23 years before retirement owing to ill-health. Gradually rose to manage bookshop but owing to ill-health his position fell until he again had to do inferior work. This upset him very much. Reserved nature, sensitive and overconscientious, ambitious to make a secure career for himself, not suspicious or jealous. Married 14 years, five children, devoted to family. *Previous illnesses:* Rheumatism and chorea at the age of seven, acne rosea eight years ago and has suffered all his life from mitral stenosis and enlarged heart. Frequently off work owing to early heart failure, or condition of his face. Corneal ulcers supervened three years ago and he has gradually been losing his eyesight. Treatment at various voluntary hospitals since 1928. *History of present illness:* Since 1928 has been unable to work efficiently owing to his illness and gave up work altogether in 1930. Long periods on and off in hospital. Christmas 1934, became depressed, thought that his family had no further use for him and might leave him and even suspected the doctors of poisoning his medicine. Complained that other patients talked about him. He also appears to have been hallucinated, both visually and aurally. Died May 25, 1935.

Case 4 (W.G., æt. 40).—Admitted August 30, 1932. *Complaint:* Sleeplessness and 'loss of will power.' *Family history:* Father committed suicide and had had asthma badly

for many years. Was subject to attacks of depression and had occasional ideas that he was being followed about. He occasionally drank alcohol immoderately. He was twice married, the patient being the offspring of his second marriage. An uncle on his mother's side died in an asylum of some unknown condition. *Personal history :* Had shingles as a child and was delicate. At 12 he had some twitching condition like St. Vitus' dance in his face and fidgetiness generally. On leaving school, where he did well, he worked on his father's farm and later took over the farm. He has since bought and sold a number of farms, always to his profit. He mortgaged his last farm, however, and disposed of it in a hurry somewhat to his detriment. He has been married 14 years and has four children, all of whom, except the first who died five years ago from meningitis, are healthy. He had brief spells of depression all his life, chiefly in morning, wearing off during the day. He has been energetic, religious, anxious and somewhat emotional. *History of present illness :* Began five months ago. He worried over his disposal of the farm, and over the illness of his children, which actually was of a transient and unimportant kind. Later he became more easily upset and had peculiar feelings in which he had the sensation of being very strong and very elated ; these would last about 10 minutes. He had also frequently been agitated and prior to his admission had been so restless that it had been necessary to give him injections in the nursing home where he was treated prior to his admission. *Condition on admission :* Apart from a facial tic there was nothing remarkable in his general aspect, but in his talk he continually expressed fears as to the wisdom of his business arrangements and their effect upon him and his family. He was preoccupied about his health and at times agitated. Sensorium clear. He was unable to recognize that his fears about his business were excessive or that he was still ill. *Progress :* He worried extremely over any slight mishaps that crop up, e.g. nightmares, slight pains in the head, but on the whole he improved until some business arrangements which his solicitor had carried through for him were regarded by him as likely to be ruinous and he again became rather agitated and insisted on leaving hospital in order that he might attend to these matters himself. Re-admitted, February 21, 1933. *Interval history :* Was worried and sleepless, often wandering round the house at night thinking he was going to be ruined owing to financial reverses, which in fact were quite unimportant. 'Nerves never stopped working ; felt screwed up in the head.' Finally started to have suicidal fantasies and felt he could no longer trust himself. *Progress :* On the whole became worse. Always agitated and extremely worried about himself. Depressed at times. Complained of an endless sequence of somatic symptoms, pains in all parts of the body, inability to sleep and eat, etc. Also feared he was losing grip on himself and would 'go mad.' Frequently demanded interviews, but could not be reassured. Finally discharged himself when he was transferred to the acute ward and went to a mental hospital.

Case 7 (Mrs. B.K., æt. 35).—Admitted January 6, 1933. *Complaint :* Confused. *Family history :* One sister suffers from chorea. *Previous history :* Early life unknown. Did well at school. At 24 was in hospital with chorea. Married at 25. Husband unemployed for a large part of the time. Four children healthy, one miscarriage. Always quiet, reserved and made few friends. Periods regular, moderate habits. No previous illnesses. *History of present illness :* Apparently since two years after marriage patient became unreliable and bad-tempered. Two years ago she became quieter and secretive. Following the birth of her baby about September 1932 patient avoided people, and thought people were looking at her. In December she told her husband that two of the children were not his. She then became restless and excited and noisy. *Condition on admission :* She is abstracted, does not reply to questions and is depressed and tremulous. In addition she shows many mannerisms and her conversation is disjointed. She says she is tortured, that people talk about her and say her children are dirty. She hears the voices of her brothers and cousins. *Physically :* Roughening of first sound at apex. W.R. negative. *Progress :* For a time patient remained quietly in bed refusing to speak and lying with her head covered. She soon became restless, said she heard patients whispering her name and

looked perplexed. She became impulsive and showed catatonic symptoms, viz. grimacing, attitudinizing, and resistiveness. She mistook identities, said the doctor was her brother, and that there were men in top hats about. She became aggressive and hostile in her attitude, gradually became more detached and on one occasion struck a nurse. Owing to the prognosis being considered bad she was discharged to another hospital. Husband still notices some signs of twitching.

Case 21 (A.Y., æt. 26).—Admitted March 31, 1932. *Complaint :* Sensations such as 'I am not here,' 'I feel as if I am not me,' 'This is not my mind.' *Family history :* Father neurotic, one maternal aunt in mental hospital (? Korsakow). *Personal history :* Normal development. No previous breakdowns. Reached top standard at school although she was away for nearly two years with chorea. St. Vitus' dance at 21. About the age of 20 she appears to have had vague neurotic symptoms. *Previous personality :* She was always nervous and jumpy, but sociable, enjoyed a good time, liked to be the centre of attention. Said to have been jealous. *On admission :* Depressed with depersonalization and some lack of vividness in appreciating her surroundings. There were in addition many hysterical features. She was shaky and her limbs trembled. She had affectations of voice and gestures, and employed many superlatives. There were no delusions and hallucinations. She made good progress and, according to a letter, has recovered since.

Case 22 (Mrs. L.F., æt. 36).—Admitted June 16, 1932. *Complaint :* Depression. *Family history :* Mother suffered from 'nerves.' One brother suffered from shellshock after the war. *Previous history :* Infancy and childhood normal. Suffered from chorea. Moderate at school. Married in 1917, had 10 children, four of whom died. Somewhat reserved, shy and sensitive. Moderate habits. *History of present illness :* Patient thought she had been sterilized in hospital and was rather upset when she found she was again pregnant. Last baby was born three months ago by Cæsarean section. Two days after operation she became depressed, apathetic, and thought people were talking about her. *Condition on admission :* Depressed, suspicious and paranoid. Weeps at times, rather vague and evasive. Thinks men are after her and that there is scandal being talked in her neighbourhood about her. *Physically :* Suffers from mitral stenosis. *Progress :* For a short time patient remained settled, but she gradually became restless, suspicious and abusive. Repeatedly attempted to run away, fought violently when an attempt was made to bring her back and refused to re-enter the hospital. Discharged not improved to another mental hospital.

Case 23 (Mrs. R.L., æt. 29).—Admitted May 21, 1932. *Complaint :* Depression. *Family history :* Sister died at 23 from cerebral hæmorrhage. *Previous history :* St. Vitus' dance at five or six years. Early history unknown. Left school at 14, standard five. Attack of neurasthenia in 1925, depressed. Attended heart hospital in 1929. Since then breathless, moderate habits. Married in 1926. Described as sensitive, easily disturbed. *History of present illness :* In May 1932 baby was born. Patient became suspicious, restless and talkative. *Condition on admission :* Resistive, talkative and rambling. Stays in bed but refuses medicine. Distractible. Thinks people are jealous of her. Eyes sunken, hectic flush. Disorientated. Incoherent. Memory impaired, and answers vague. Mitral stenosis. *Progress :* Patient showed no change after admission. Remained suspicious, restless, talkative, agitated, disconnected, restless at night. Refused food and medicine. Appeared terrified. Husband took patient home not improved against advice.

Case 24 (Miss L.M., æt. 19).—Admitted September 19, 1933. *Complaint :* Chorea accompanied by screaming attacks since July 1933. *Family history :* Patient is the seventh of 11 children, in poor circumstances. Family history negative except for maternal grandmother who became 'senile.' *Personal history :* Early childhood was uneventful apart from rheumatic fever which occurred for the first time at 14, recurring at 16½. On the second occasion it was accompanied by chorea which she has had off and on ever since. *History of present illness :* Before 13 normal, getting on fairly well with people although quarrels with other children. Between 13 and 16 energetic and sociable, fond of parties, lots of friends.

Since rheumatic fever difficulties at home, criticizes people. Swings of mood. Very clean, afraid people may criticize her. Never sits still. Fidgety with arm and legs; movements of choreic type. At times large and spasmodic movements. In July 1933 was in hospital with rheumatic heart and chorea; while there, had screaming attacks, one of which lasted on and off for three days. She became somewhat excitable and asked to see her mother, who had died some months previously; complained of terrifying dreams. *Condition on admission:* Physical condition not satisfactory. She had an enlarged heart, mitral regurgitation and typical movements of chorea. Mental state: not cooperative and refused to talk. She appeared preoccupied but it was impossible to get any indication of her mental condition. There was some patchy anæsthesia of the left side of body. She denied any memory of her screaming attacks. *Progress:* A few days after admission she started to scream again, became dazed and 'far away' in between the bouts of screaming. Her condition became so bad that treatment in the acute ward was required. While there her state varied; at times she became friendly and cooperative, this was usually associated with improvement in the chorea. At other times she would be petulant and making complaints of the treatment. Her choreiform movements would cease abruptly and it was felt that her condition was partly choreiform and partly hysterical. She improved gradually and was finally discharged improved. She has had several such recurrences. Final condition moderately satisfactory.

THE SIGNIFICANCE OF THE FRONTAL LOBES FOR MENTAL PERFORMANCES*

By

KURT GOLDSTEIN, New York

Opinion concerning the significance of the frontal lobes for psychic performance is divided. Nobody, however, will nowadays deny it some significance, as was the case not very long ago. Clinical and experimental facts have furnished too obvious a tale for that. It is not my intention to review here the pertinent literature or to discuss the interpretations proposed by the various workers in this field. I shall confine myself to setting forth the facts emerging from my own investigations. But before I do so, I would briefly touch on the grounds underlying the prevailing divergence of opinion.

There are various groups of such causes. (*a*) Inadequate attention has been paid to site, extent and nature of the lesion. Cases of small circumscribed tumours or injuries need not show any mental disturbance, particularly not under examination by the usual methods, and more especially when the lesion is in the right half of the brain. Ascertainable mental disturbances will be found only in cases of extensive lesion localized in the prefrontal region in both hemispheres, or, at least, an extensive lesion in one hemisphere, especially the left. It is not possible, however, to generalize on the appearance of psychic symptoms in lesions of the left side, because the so-called cerebral dominance of one hemisphere is subject to individual disposition and no hard-and-fast rule can therefore be given. It is important also to pay attention to the nature of the lesion, because in different diseases the amount of microscopic tissue damage extending beyond the gross lesion may differ widely.

(*b*) Another factor leading to differences of opinion lies in the differences in manner of investigation. As I shall attempt to set forth later, the psychic changes characteristic of lesions of the frontal lobe are of a definite type and may easily be overlooked, because the methods of examination usually employed are unsuited for disclosing them. The psyche may appear undisturbed because the patient's behaviour may be ostensibly normal, in spite of definite psychic alterations, the existence of which can be discovered

---

* Read at the Second International Neurological Congress, London, 1935. From Columbia University and the Montefiore Hospital, New York City.

only by special examination. In general it is to be said that negative results must be regarded as of less importance than positive results, particularly as the considerations mentioned previously have received insufficient or no attention.

For these reasons, the greater part of the literature is useless for answering the question whether psychic disturbances occur in cases of lesion of the frontal lobe, and if so, what these disturbances are. My personal opinion as to the importance of the frontal lobes for mental performances is based, in the main, upon my own investigations of a great number of cases of both injury and disease, most of them kept under observation for a period of years. For the methods of investigation employed, I refer to my earlier publications. My view of the psychic function of the frontal lobe is set forth in a paper published in 1923.[1] Subsequent investigation confirmed the earlier findings. In addition I would mention two papers by Brickner [2] and Penfield,[3] published recently, with instructive observations on patients who had undergone surgical operation on the frontal lobe. I shall revert to them later. With the experiments on animals by Fulton, Jacobsen and others, I cannot deal here, but should like to mention that their results appear to agree with mine.

SYMPTOMS OF MENTAL CHANGE

Now, let me proceed to sketch some of the phenomena of mental change to be found in patients with lesions of the frontal lobes (cf. Siekmann[4]).

Here is a man, 30 years of age, who does not seem to be very much disturbed in his customary way of life. He is a little slow; his face is rather immobile, rather rigid; his attention is directed strictly to what he is doing at the moment—say, writing a letter. Addressed, he will be somewhat startled, and give the impression of one awakened from sleep. He first looks at me quite astonished, then he smiles and gives the necessary replies or performs the necessary tasks, if we set him tasks which he is able to perform. As to the tasks which this patient can perform, and those he cannot, there seems to be, at first sight, no regularity that would make it possible to speak of his showing a lack of capacity for any specific performances. In fact, it would seem at times as if the patient had no defect at all in performance, except perhaps a degree of slowness. He seems to be oriented in time, space, and the attributes of his person. He gives correctly the date of his birth, the ages of his children, etc. He can count, and is able to solve simple problems in arithmetic. He can reproduce the elements of his schooling and can report the events of his more recent experiences.

I tell him a simple story, and he is able to recount it after some interval. He can draw simple designs, a house or human face, can copy a simple drawing, and recognizes pictures known to him. His language is somewhat simple in structure. Frequently he seems to be hunting for words, but when the right word is supplied he usually recognizes and adopts it readily. He can read

and write. He does very well in simple reaction tests—for instance, in a test requiring him to withdraw his finger from a reaction key whenever a light is shown. Whatever fields of performance I tested, and I tested a great many, I may say that in nearly all I found apparently normal responses. As I have said before, the patient is slow; at times he appears to make a great effort to respond; he perseverates; yet, up to this point, he has shown no grave defects.

But, in the same patient we may also encounter *a totally different behaviour*. In tasks which on the surface seem closely similar to those he has performed quite well, *he suddenly fails completely*.

The patient has just copied successfully a little ' house ' built up of small wooden sticks. Now, we place before him one of these sticks in a definite position, pointing, for example, from left below to right above. He is asked to note well the position of the stick. After a half minute exposure the stick is removed, then given to the patient; he is asked to put it in the position in which he had seen it before. He tries to replace the stick, but fumbles. He is all confusion, looks at the examiner, shakes his head, tries this way and that, plainly uncertain as to the correct response. The final upshot is that he cannot replace the stick in the required position. The same inability we may observe if we put before the patient two sticks placed together so as to form an angle with the opening pointing upward. The patient tries to put the sticks together in the same way, but is unable to place them in the proper position. If he succeeds it is manifestly accidental. Next, we show the patient a little ' house ' built up of such sticks, a house with a roof, a door, a window and a chimney, and he succeeds very well in reproducing the design.

At first sight, these differences may seem totally ununderstandable. Further examination clarifies the situation. We have seen that the patient fails in the test with two sticks joined together in the form of an angle with the opening upward. It appears amazing at first that if we compose the same angle with the opening pointing down, the patient will reproduce the figure very well at the initial trial. When we try to ascertain how this is possible, when we ask the patient how it happens that he can reproduce the second figure but not the first, he says, ' this one has nothing to do with the other one,' and ' this is a roof.'

These two replies lead us not only to an understanding of the patient's behaviour in these tests but also of the fundamental change undergone by him.

What is the difference between the two tests ? What produces effects so contrary or what accounts for the differences of the behaviour of the patient in performing these tasks ? His first reply makes it clear that the two objects with which he has to deal in these two tests are to him totally different from one another. The second answer shows that the angle pointing downward is apprehended by the patient as a concrete object of his own visual experience, and he constructs a concrete thing with the two sticks. A concrete apprehen-

sion and concrete behavioural action are sufficient to meet the conditions of this test. In the former test the two sticks did not arouse in the patient an impression of a concrete thing. He has to conceive of the positions of two meaningless sticks in a meaningless connexion, one with the other. He must regard the sticks as mere representations indicating directions in abstract space. Furthermore, he must keep these directions in mind and rearrange from memory the sticks as representatives of such abstract directions.

In the first test the patient needs to deal simply with a known object; in the second one he must give an account to himself of relations in space, and act on the basis of abstract ideas. His action is not determined directly by a given concrete thing, but by a representative of an abstraction.

The disturbance of the patient, it is apparent, lies in the circumstance that he is unable to assume an attitude towards the abstract, but is able to act in a concrete way. Therefore, he is unable to perform tests the execution of which demands that attitude. The angle test with the opening pointing down does not demand it and the patient is able to execute it perfectly. It is for the same reason that he is unable to place a stick in a definite direction but is well able to imitate the constructing of the little 'house' which does seem to be much more complicated.

(We may conclude from these slight indications that the patient's lack consists, in these tasks, in an incapacity for a definite behaviour which we may call abstract behaviour.) We shall soon reach a more precise characterization.

METHODS OF EXAMINATION

Let me interpose here a few words bearing on the method governing the examination. If it happens that the patient has given to him only tasks of the second kind, for example, the angle which he identified as a roof, the examiner will obtain the impression that the patient's spatial attitude is perfectly normal. His failure—in face of the singularity of the task of the other kind—will then be readily attributed to other causes, such as inattentiveness, fatigue, etc., as long as one has not recognized the specific difference of the two sorts of performances for the patient. This difference may be easily overlooked, since to a normal person there is no real difference. In the same way, one can be wholly deceived concerning a patient's spatial competence, by noting that he can quite correctly point to the source of noises or can lead us, on demand, from one place to another over an apparently complicated path. That, none the less, this patient suffers from a grave spatial disturbance becomes manifest as soon as we simply ask him to tell where the noise is, instead of pointing to it, if it is on the right side or the left side—or to describe the path over which he has just conducted us. Then he is at his wit's end.

The same is to be observed when we ask him to traverse the path from a different starting point. In the latter case he can no longer support himself by the concretely known path, but must give an account to himself of the situation from a new starting point, with its relation to the goal and to the

possibility of reaching it. Because he is unable to do so, he fails. An example: A female patient, suffering from such a disturbance, led us from the examination room, well known to her, to her ward and her bedside, by the shortest possible route. In the same way, she later took us from the ward to her workroom, situated several stories higher. She went to the elevator without the slightest hesitation, pushed the right button and arriving at the workroom floor, led us to her place there. The entire trip was quite complicated. She would make the return trip from the workplace to her ward in the same unerring manner. But when she was led into the corridor on the workroom floor and then asked to take us to her ward, she went up the corridor, which is a replica of the one on the ward floor, just as if she were actually in the latter. Naturally she reached the wrong place, noticed it and showed signs of annoyance. She had no inkling of how she had come to a wrong place, could not understand where she was, and was unable to find any way out. Her helplessness was due to the fact that it was impossible for her to conceive on what floor she was. This behaviour makes it clear that her correct routine accomplishment of this trip between ward and workplace did not rest upon any real knowledge of the path but upon simple concrete behavioural action. If this distinction is not heeded, a gross misjudgment of the patient's spatial attitude may result from his ability to go from one place to another in a complicated passage.

These examples show that it depends entirely upon the nature of examination whether we achieve a correct judgment as to a patient's capacity or reach false assumptions about his disturbance.

SPATIAL AND TEMPORAL DEFECTS

Our assumption concerning the defect in the patient finds its confirmation in examinations of many and varying performances, of which I can give only few examples.

First, in a test in which the apprehension of space relations plays a great rôle, the patient is to throw a ball into boxes successively situated at a distance of 3, 9, 15 feet from him. He does it quite well. Then he is asked how far the several boxes are from him, but he is not only unable to answer this question, but even to say which box is nearer, which farther. However, he may give correct replies if he is allowed to walk over a definite distance and to count his steps, knowing that each step covers 1 foot. He can tell by counting how many feet there are for each distance and thus can infer as to which box is farther and which nearer.

What is the difference between the two tests? In the first, the patient has only to deal with objects in a behavioural fashion. It is unnecessary for the patient to be conscious of his act and of objects in a world separated from himself. In the second test, however, he has to separate himself from the world and must give himself an account of his actions and of the space relations in the world facing him. Therefore he fails.

Now, let us regard the behaviour of the patient in situations in which space does not play an essential rôle.

We find in our patients the same modification in their attitude to time as to space; that is, they can tell us certain aspects of temporal things but they really do not know what they mean. They cannot really distinguish between different durations. Actually they do not understand the meaning of longer and shorter time. Their concrete behaviour indeed might arouse the impression that they are quite at home in matters of chronology. An example: One patient was required to present himself for repeated examination at four o'clock. He had a journey of three-quarters of an hour from his house to the hospital. He always arrived with the greatest punctuality. How did he manage it? He knew it would take him 45 minutes. He knew that in order to be at the hospital at four o'clock he must leave his house at a quarter past three. These factors were furnished to him by a certain position of the hands of his watch, without his knowing or needing to take into account their meaning in a general way.

Though such patients show their ability to use a watch apparently normally, none the less they have no sense of time at all. This is brought about by a simple test such as asking them to say where the minute hand of a clock is at a certain time, for example, at 13 minutes past four o'clock. A normal person would immediately say: slightly ahead of the hour-figure two. Our patients, on the contrary, could not give a description at all, or could reach it only when they are allowed to point with their finger at the minutes from 60 to 13.

OTHER FAULTY REACTIONS

Now, let us observe the behaviour of the patient in situations in which space and time do not play an essential rôle.

We choose a simple reaction test: the patient is instructed to execute a simple movement in response to an abruptly flashed signal. After some practice he learns the situation. He reacts correctly in relatively short time. We then flash a red light, then a blue light, and the patient is instructed to execute the movement on seeing the red light but to do nothing on seeing the blue. In this and similar selective reactions his performance is inadequate. He seems to become confused and either does not react at all or makes many errors or reacts after long intervals.

What is the difference between the two tests? In one the patient has to react in a simple way to a simple stimulus. His behaviour is simple, directly determined by the stimulus. In the second test he has to choose: that means, he has to face two possibilities, or, in other words, an abstract situation, and here he is defective because he cannot achieve such an attitude. This is the very thing he cannot do because he is unable to deal with any merely 'possible' situation at all. Thus we can describe the deficiency in this patient as a lack of capacity for approaching a 'possible' situation.

The following test, in quite a different field, adduces confirmation. A simple story is read to the patient. He seems unable to understand it. Perhaps he repeats some single words, but he does not understand their meaning and is unable to repeat the essential points of the story. Now we read him another story which for a normal person is surely not easier to understand. This time the patient understands the meaning very well and recounts well the chief points.

What was the difference between the two stories? The first one dealt with a simple situation, but a situation which had no connexion with the actual environment of the patient. The second story, on the contrary, had a direct bearing on his situation. Again we observe that the failure is due to incapacity for an abstract approach, or, as we may also express it, an approach to a situation presented only in imagination. Choosing stories with this point of view, we are able to predict beforehand which ones the patient will be able to understand and which not.

The same lack is observable in tests with graphic representations. Pictures of single objects are recognized almost always. In illustrations which contain a number of things and persons in internal contact with each other, the patient may pick out some details, but is unable to understand the picture as a whole and fails in acting in response to the whole. A precise examination reveals that the patient's real understanding does not depend on the greater or smaller number of components in a picture, but on whether the components, whatever their number, hang together concretely, and in ways familiar to the patient's experience, or whether understanding of their connexion requires a more abstract synthesis on his part. In one case, the patient may apprehend pictures with many details. In the second, he may lack understanding even if there are only few details. The patient's deficiency lies in his inability to find out the essence of the picture. Thus, I may also characterize his total deficiency as merely another manifestation of his unfitness for finding out the essence of any situation.

If the patient is given enough time he may, after recognizing some details, then draw a conclusion which enables him to react with passable correctness. Since in tachistoscopic examinations the time is too short, the patient fails in such examinations. He fails also if the test demands the comprehension of a succession in time of separately given parts of a situation, as, for example, in picture series (such as the comic strips of American newspapers) which cumulatively tell a story.

In the examples mentioned, we have already seen the change in the patient's capacities for action. More definite examinations of his actions disclose the same characteristics. It depends on the situation whether the patient is or is not able to execute two acts apparently equivalent. Differences between the two acts insignificant to normal apprehension may suffice for his succeeding in one act and failing in the other. Failure is the more certain and pronounced if, after he has begun to act, the situation is modified in

such a way as to require readjustment on his part. Such a change in adjustment or approach is an out-and-out impossibility for the patient. Therefore, he is wholly unable to continue an action thoroughly familiar to him when it is suddenly interrupted by outside interference. Let us suppose he is made to count from 1 to 20: interrupt him when he comes to 9, and he cannot continue. He may be able to write a phrase or a sentence, but he is unable to continue if he is interrupted during the writing, and so on in other activities. In such situations there develop partial reactions, false position of the parts, perseveration, etc. Acting may be further altered by deficiency in emotion and memory, of which we shall soon speak.

In situations which do not allow correct reactions, the hindrance to action may become so great that the patient becomes excited or confused and loses interest in trying to place himself in relation to his environment, because he cannot do so effectively and these attempts only throw him into fresh confusion. The result is so-called 'akinesis'. The tendency to refrain from action may be increased by motor difficulties, such as change of tone, disturbance of associated movements, adjustment, movements, etc., which often occur in frontal lobe lesions. The abnormal difficulty of all behavioural action causes the patient to abandon each and every activity and leaves him also appearing affected much more in his mentality than is really the case.

Some authors have also spoken in this connexion of an impairment of a primary impulse drive, of volition. Such terms do not correspond to the real facts. The 'will,' the 'Antrieb' are not disturbed at all, but they cannot function, because to many situations in which 'will' appears the patient is unadjustable. The patient appears disturbed in will and 'Antrieb' in general.

EMOTIONAL CHANGE

Frequently the patient exhibits a dulling of the emotions, but we note also in other situations that the same patient does not appear to be without feeling. On the contrary, we observe in him a great excitability. These facts make it doubtful whether there is really a simple emotional defect. I do not suppose so. If we analyse precisely the situations in which the patient appears emotionally inert and those in which the contrary is the case, we ascertain that the presence or absence of emotional expression corresponds to his entire behaviour in the given situation, and that the emotional behaviour is at best inseparable from the rest of the behaviour. The fact seems to be this: If a patient apparently does not react emotionally in a satisfactory way, it is in situations in which he also fails to comprehend the essentials to which a definite feeling attaches. The patient appears emotionally inadequate when he is in a situation in which that feeling which we regard as belonging to it cannot be aroused because the patient may have grasped only a part of the situation to which that affect which the patient shows may be appropriate. It appears to us inappropriate because we regard the whole situation and not merely a part of it. If we regard the behaviour

of the patient from this point of view his feeling does not appear abnormal to the situation as it is experienced by the patient.

Thus we have to understand the lack of interest in the next example, in a patient with a large lesion of the frontal lobe. This patient, a male, never seemed to be concerned about his family. He never spoke of his wife or children, was irresponsive when we questioned him about them and when it was suggested to him that he should write to his family he would show cool indifference. Thus he appeared to lack all feeling. Now it was an established practice that he should visit his home, situated in another town, and stay there several days. While at home the patient conducted himself, as we learned, quite as would a normal man in the bosom of his family, kind and affectionate to his wife and children, interested in their affairs, in so far as his ability would permit. After his return to the hospital from such a visit and being asked about his people, he would smile in an embarrassed way, giving evasive answers. He seemed utterly estranged from his home situation. Unquestionably what ailed this man was not really a deterioration of his character on the emotional and moral side.

Our assumptions also lead to an understanding of the fact that the same patient who appears very dull may suddenly become excited in a situation which at first seems to contain no cause of irritation. Analysis makes it intelligible to the patient that this situation is of such a nature to give cause, from his point of view, for becoming excited. This, once recognized, is but a small step to a recognition of the fact that the—to us—groundless excitement of the patient is—to him—perfectly rational. An example: A patient of mine had a friend who was his close companion. One day the friend went to a cinema with another man. He did not take our patient because the latter had seen the particular picture before and would not go to see it a second time. When the friend came back from the ' movie ' our patient was in a state of great excitement and refused to speak to him. He was not to be quieted by any arguments. No explanation that his friend did not want to offend him, that his friendship had not changed, made any impression. From that time on, our patient was his old friend's enemy.

This reaction, at first so unintelligible, can be understood if we remember that the patient is able to make only a direct concrete approach to any situation. This is also the case in his approach to his friend. He saw only that his friend was keeping company with another man and he felt himself slighted. He was unable to understand that his friend's conduct in no way actually affected their relations. He could not recognize why his friend went without him and he could not perceive the situation as a whole because he could not abstract it from his own concrete personality. He saw only the concrete separation between himself and his friend and from this standpoint his exaggeration was thoroughly understandable, particularly if we consider how difficult it is, in the case of a change of attitude, for a patient with a lesion of the frontal lobe to enter into the relation of friendship. The patient

felt his loneliness and sank into a catastrophic situation of confusion and anxiety. He regarded his friend as its cause. This insight into the workings of his mind renders his behaviour intelligible. Since many situations are not grasped conceptually by the patient, his total affective state must necessarily be more equable and the result is that he appears emotionally blunted.

The mania for inane witticisms, so often described in our patients, finds its explanation in a similar manner. Their jokes, to us so meaningless, do not spring from an abnormal feeling for relations—but from the appeal to them of some details in a situation which we normal people do not notice because we see the situation as a whole. Once that detail comes to our attention, we discover that it is really of a nature to arouse our own sense of humour too. The mania for witticisms is thus seen to be but another manifestation of the patient's changed attitude toward the world.

MEMORY AND ATTENTION

Finally, we have yet to deal with two symptoms very frequent in frontal lobe lesions, viz. alteration of memory and of attention, which are often considered the basic disturbances in these lesions.

Considering memory first, we find that under certain circumstances the faculty for reproduction of facts acquired long ago may be normal. For example, school learning, etc., may be recalled very well in some situations—but not in all. The situation must be adapted to reawaken old impressions. The patient must be able to regard the present situation in such a way that facts of the past belong to it. If that is not the case, the patient is unable to recall at all the identical facts which he has reproduced very well in another situation.

He is incapable of recollection when he is asked to recall things that have nothing to do with the given situation. But when it is possible to put him into a situation to which the material inquired for belongs, recollection appears suddenly. The patient is also unable to remember if the required answer demands an abstract attitude or whether it demands that he give an account of the matter in question. Therefore, the patient fails in many intelligence tests which may seem for us very simple—and he is amazingly successful in apparently difficult ones.

Observation of the patient in other situations demonstrates clearly that memory failures are not caused by an impairment of memory content but that the lack of memory is caused by his faulty attitude or approach that is requisite for the specific test. The patient has the material in his memory but is unable to use it freely; he can use it only in connexion with a definite concrete situation to which it must seem to him to belong. In the same way he is able to learn new facts; he may be able to learn numbers, syllables, or movements by heart; he is able to hold in memory situations, facts of environment, etc., but he is able to learn these only in a concrete situation and to reproduce them only in the same situation in which he has learned them. In the specific situation, he may be very well oriented, but he is

unable to give an account of some place he had visited. Therefore, the acquisition of new material remains scanty.

Thus we come to the conclusion that there is in these patients not a real defect in remembering, but that reproduction and new acquisition are defective because the normal basis of reproduction and acquisition is not given. Therefore the patient's performance constantly varies according as the task is embedded in a concrete or in an abstract situation. In the first, the patient performs well; in the second, he fails. Since in normal memory the abstract situation plays an important rôle, for reproduction as for acquisition, the memory of the patient seems to be feeble.

Finally, as to attention: Here, too, one finds wide differences in the facts. At one time the patient appears inattentive, at another even abnormally fixed in attention. Attention is usually weak in the special examinations, particularly at the beginning of them, before the patient has gained the real approach to the whole situation. In such a situation he appears much distracted. However, if the patient comes into the picture, then his attention may be satisfactory, may even be abnormally fixed. He may be totally untouched by other stimuli from the environment to which normal persons will react unfailingly. Thus he may appear distracted in some tests, in others attentive, depending upon whether he is equal to the test or not. In some tests he will always appear distracted, for example, in those which demand a change of approach (a choice reaction) because he is incapable of making such a reaction. Thus it is not correct to speak of a change of attention in these cases in the sense of a plus or minus of attention. The patient's state of attention is but an expression of his total behaviour and is to be understood only in connexion with it.

DISCUSSION AND EXPLANATION

Summarizing the facts found in cases of frontal lobe lesion, we may say: (1) There are disturbances in different fields of psychic performance, in each field perhaps, but the disturbance of no one field can be regarded as the basic cause of the other disturbances. (2) No field is disturbed throughout its extent. There are always some performances which the patient is able to execute. (3) The basic change is not a change of any one field or any one performance, but a change of total behaviour, with a lack of a particular behaviour, which lack finds expression in certain performances in all fields and leaves intact certain performances in all fields.

We have given only a few examples from a few fields. More comprehensive investigation including diverse fields discloses the same state of affairs. From our observations, it can be said that all performances are changed in the same way: thinking, acting, feeling, attention, volition, counting, speaking, understanding, orientation in space, in time and all other factors which we may examine.

I have characterized the patient's lack in different terms, as lack of

abstract attitude, as lack of approach to imagined things, as inability to give himself an account of acting or thinking, as inability to make a separation between the ego and the world. At bottom, all these terms and others which one may use to characterize the facts mean the same. The widely different manifestations in behaviour represent, without exception, one and the same defect in the underlying attitude.

We can distinguish two different kinds of human attitude toward the world: (1) A *concrete* attitude, in which we are directed toward given objects and directed in our thinking and acting by them; (2) a more *abstract* attitude, in which we are moved at first to think about the objects and to give an account of them to ourselves. To each of these attitudes belongs a particular kind of behaviour. In the first we are acting in the world, manipulating the objects; our activity is determined directly by the claims of the objects upon us. In the second attitude we are thinking rather than acting, even our activity being directed by thinking. Our actions are governed not so much by the objects before us as by what we think about them. In the first attitude we behave more passively, in the second, more actively. Some tasks can be performed only by the one type of behaviour, others only by the other. In patients with lesions of the frontal lobe, active (abstract) behaviour is lacking, but the concrete behaviour may be very well preserved.

From the standpoint of general physiology the patient's deficiency may be designated as a disintegration of a higher, more complicated process, which is rather determined by the whole organism, to a less complicated, lower one, determined more by the stimuli of the outer world. This disintegration of function is well in accord with the general type of disintegration of the function of the cortex, wherein all of its varieties exhibit the same ground form.[5]

This proposition, in general, well meets the conception of 'dissolution' put forward by Hughlings Jackson. Here I do not wish to deal with some disagreement that exists between the opinion of this great master of neuropathology and my own, a difference which concerns his contention that disintegration, or as I would prefer to call it, 'dedifferentiation,' means a 'dissolution' which he regarded from the standpoint of evolution.

Here I should prefer to emphasize what conception Jackson and myself have in common, as such agreement is much more important than are any differences between us.

Concerning the facts at issue, a number of more recent observations, particularly those of Penfield and Brickner, agree with my own. This agreement carries particular weight because the cases of these authors deal with lesions of the most unambiguous character, each involving surgical operation and anatomical examination.

As far as I can gather from the case-reports, the symptomatology differs markedly from the symptomatology I have described. But the difference is only an apparent one: viewed more closely, the differences are seen to be

the result of a variation in methods of investigation. If the phenomena described by the authors are re-analysed from the point of view set forth here, they would lead to a conception of the basic changes identical with mine.

As to these authors' views concerning the psychic deficiency in the patients observed by them, Penfield contents himself with mere matter-of-fact. Brickner reaches a conception which on some points is in agreement with mine, but in others not. He, as I do, rejects the notion of alterations of different single mental performances, such as memory, intelligence, affect, etc., but assumes a basic deficiency, as expressions of which the different disturbances are to be regarded. I should like to stress particularly that we agree that the emotional symptoms act simply as an expression of this basic change, not as an affection of any particular function. I emphasized this view years ago.

I should not omit to mention, however, that despite this general agreement, I differ from Brickner in the conception of what is the basic change. I contend that there is not only a lack of synthesis, as Brickner assumes, but a lack of a definite form of general behaviour of which the patient's inability to ' synthesize ' is only the consequence, or more correctly, a phenomenological expression of it. Synthesizing, it should be remembered, is not a primary psychic function, but a performance, and like all other performances it may appear disturbed or unaffected, depending upon whether a concrete or an abstract synthesis is to be performed. The patient's apperception may be equal to the task of ' synthesizing ' an indefinite (and quite a considerable) number of ' parts '—more exactly, what the normal observer views as parts—into a whole and to react to this whole correctly or normally, provided that the ' parts ' will fit together for him concretely. If they do not fit together concretely to satisfy him he fails.

The facts here displayed furnish the foundation for the diagnosis of affections of the frontal lobe. It is true that the modification is not always of such an advanced form as that which we have described. According to our view that performances differ in point of complexity or simplicity, varying degrees of lesions will entail varying degrees of impairment of performance. Important for the diagnosis is the establishment of the particular nature of the change undergone by the patient's performances. If the examination is undertaken in the proper way and if the damage is not too slight, the characteristic modification will, without doubt, be discoverable in some examples of performance.

In attempting to base the diagnosis of a frontal lobe lesion on the mental change there is one differential diagnostic difficulty—the determination of a frontal lobe lesion as against a general damage of the entire cortex. There is no question but that the clinical picture of the mental change is very similar in both affections, and many investigators tend to assume that the mental disturbances in frontal lobe lesions are conditioned by such a concomitant general affection. The facts warrant no such assumption. We have enough

trustworthy observations of definitely circumscribed lesions of the frontal lobes which exhibited all of the characteristic symptoms.

Why then the similarity between these cases and diffuse affections? The function disturbed in either case is one of the highest order, making the greatest demands upon the cortex. Owing to the damage of a certain degree of severity the highest function will naturally be the foremost to be disturbed. The frontal lobe is essential for the highest functions. Therefore we find identical symptoms in lesions of the frontal lobe and in diffuse cortical affections which implicate always, as well, the frontal lobe. The symptoms observed in cases of frontal lobe lesions are therefore not manifestations of an assumed general cortical affection; on the contrary, the characteristic symptoms in diffuse cortical affections are manifestations of the damage to the frontal lobe. A close consideration of the whole—psychic and somatic—picture will make it possible to decide if the psychic change, as mentioned, is caused by a localized process of the frontal lobe or a diffuse lesion of the brain cortex.

Finally, I wish to stress one point: The significance of the frontal lobe for the psyche can be fully grasped only if we bring the psychic performances into connexion with those performances which we include within the function of the frontal lobe, on the ground of the determination of lesions of the frontal lobes found in such disturbances. Such performances comprise on one hand a complex of somatic processes serving the maintenance of the equilibrium of the body as a whole and of its parts, especially control of direction; and, on the other hand, certain motor activities that are internally connected with the psyche, such as speaking, writing, making gestures and the like.

From a comprehensive point of view, all these performances reveal themselves as the several expressions of a specific behaviour characteristic for the human attitude toward the world and the means of maintaining this behaviour, a behaviour which manifests itself not only in the abstract attitude, but also by the capacity for maintaining a deliberate direction of thought and movement which may be no less manifested in speaking and working and in the erect carriage.

It seems to me that the conception outlined here, perforce summarily and rather sketchily (I have developed it more fully elsewhere), furnishes the foundation for reaching an understanding of the specific place of the frontal lobe in the animal kingdom and its special development in man.

REFERENCES

[1] GOLDSTEIN, K., *Med. Klinik.*, 1923, nos. 28 and 29.

[2] BRICKNER, R. M., *Proceedings of the Assoc. for Research in Neur. and Ment. Dis.*, 1934, **13**, 259.

[3] PENFIELD AND EVANS, *ibid.*, 382.

[4] SIEKMANN, *Psycholog. Forschung*, 1932, **16**.

[5] GOLDSTEIN, K., 'Lokalisation in der Grosshirnrinde,' *Handb. f. norm. u. pathol. Physiologie.*, **10**, 600.

SPEECH PERSEVERATION AND ASTASIA-ABASIA FOLLOWING CARBON MONOXIDE INTOXICATION*

By

LOUIS H. COHEN, Worcester, Mass.

The following case is presented because of its apparent rarity, and also because of the light which it may throw upon some psychological aspects of basal ganglion function. A strikingly similar case has been recorded by Wolff [1] under the title of 'A Case of Astasia-Abasia and Speech Perseveration Following Carbon Monoxide Poisoning.' Oppenheim [2] refers to the occurrence of astasia-abasia as a manifestation of carbon monoxide intoxication. The possible significance of this case with respect to basal ganglion function is, however, our main concern in this report.

CASE HISTORY †

J.W. is a white Jewish male, 46 years of age, who attempted suicide by inhaling the exhaust from his automobile on June 18, 1935. After a four weeks' stay in a general hospital he was admitted to the Worcester State Hospital. It was reported from the general hospital that he had been in coma for 72 hours after admission, and that his neurological and mental condition had remained unchanged from that time until the day of his transfer. During this time he was said to have been 'confused and demented.'

Family History.—The patient's father died at 74, the cause of death being unknown. His mother deserted the family when the children were very young. Their upbringing was soon entrusted to a stepmother. The oldest brother, now 54 years of age, is said to have been mute since birth and is in an institution. A sister, age 52, was admitted to the Worcester State Hospital in 1929 for attempted suicide. She was considered to be suffering from manic-depressive psychosis, was depressed, and was discharged several months later, unimproved, to another institution.

Personal History.—The patient was born in New York; the details of his birth are unknown. At the age of nine he had to go to work as a peddler. He was discriminated against by his stepmother, who favoured her own children, and at the age of 17 he ran away from home to join the army. He married at 26. There have been seven children, ranging in age from eight to 19. He never had any serious illnesses. His wife has stated that he never liked to have his children near him, that he was very shy, and that he had few acquaintances and no friends. When at home he always stayed

---

* From the Research Service of the Worcester State Hospital.

† The patient, while in the Worcester State Hospital, was under the care of Dr. Morris C. Beckwitt, to whom the writer is indebted for data in the history and for a report on many of the clinical manifestations.

by himself, spending his time reading newspapers and magazines. For the past 12 years he had been employed as a caretaker on a large estate at a moderate wage. Following two large wage cuts within the past year, and after his oldest son had lost his job, he is said to have become very depressed and even more seclusive than before. On June 18, 1935, he was found unconscious in his car. There was a rubber extension from the exhaust pipe into the car; the motor was running and the windows were shut. His condition at the general hospital has already been described.

Physical examination at the time of his admission to the Worcester State Hospital on July 18, 1935, revealed a well-built male with good muscular development. His complexion was ruddy. His pupils were equal, regular, and reacted swiftly to light and accommodation. The tongue protruded in the midline with a coarse tremor. There were no unusual findings in his chest and abdomen. He was unable to support his weight on his legs, which were somewhat weak. There was no impairment of strength in his upper extremities, on which he relied to support himself. When he placed his feet on the floor they underwent marked clonic contractions. There was a coarse rhythmic tremor of the hands. The triceps reflexes were diminished but all other deep reflexes were hyperactive. The abdominal reflexes were lively. No pathological reflexes were elicited. There was continual urinary incontinence. Blood counts and urinalyses were within normal limits. The blood Hinton test was negative.

Mental examination revealed the patient to be restless, somewhat excited, and apprehensive. He was confused and completely disoriented. There were profound memory disturbances. He seemed quite apathetic, was very distractible, and showed no insight. His speech was slurred, monotonous, and rapid. When asked questions he appeared to comprehend them, but his answers were merely repetitions of the last word or two of the question. For example, when asked, ' Why did you come here ? ' he kept repeating, ' Come here, come here, come here,' etc., until he was interrupted by the next question.

A few days later the nature of his speech responses was somewhat more complex in that they now were of the nature of answers. For example, when asked, ' Did you take gas ? ' he answered, ' I took gas, I took gas, I took gas,' etc. Question : ' What kind of gas ? ' Answer : ' Regular gas, regular gas, regular gas,' etc. Occasionally there were spontaneous utterances which consisted of various requests which he repeated over and over ; for example, ' Give me milk, give me milk,' etc. ; ' Come here, come here, come here,' etc. It was noted that the tremor of both hands disappeared when he was silent but became active whenever he spoke.

About a week later his mood become somewhat more cheerful and he frequently laughed in a rather silly fashion at some of the vulgar expressions of one of the disturbed patients in the ward. He sometimes repeated these words over and over. When silent his face appeared almost mask-like. He made frequent attempts to leave the bed and appeared anxious when he was restrained. His attempts to get out of bed, however, were numerous and when successful he fell to the floor in a heap. On one occasion at about this time he said that he was 14 years old and that his oldest son was 19 years old. When asked how this was possible he answered, ' I guess it's a mistake, I guess it's a mistake,' etc., for two minutes ; his attention was then apparently distracted by some exclamation of another patient, and he said, ' Did you hear that, doc. ? Did you hear that, doc. ? ' etc. He repeated the digits 4–6–9–2 correctly, but when asked to repeat 2–9–6–3–4, for example, his response was 2–9–5–3–3–3–3–3– etc. He was able to read a simple paragraph (the Cowboy Story in Cheney's *Outline*), but mispronounced many words and left out many short words. When asked what the story was about, he answered over and over, ' It's about a cowboy, it's about a cowboy,' etc. He was asked, ' Can't you stop repeating ? ' and he answered, ' Yes, I can stop repeating ; yes, I can stop repeating,' etc., for almost three minutes. His writing at first was merely a series of irregular lines, but later was of

the same nature as his repetitive speech (see figs. 1 and 2). No tremor was noticeable while he was holding the pen. At no time was any evidence of hallucinations or delusions elicited.

During the first month of hospitalization there was a rather gradual diminution

HANDWRITING SPECIMENS

1. Write your address.

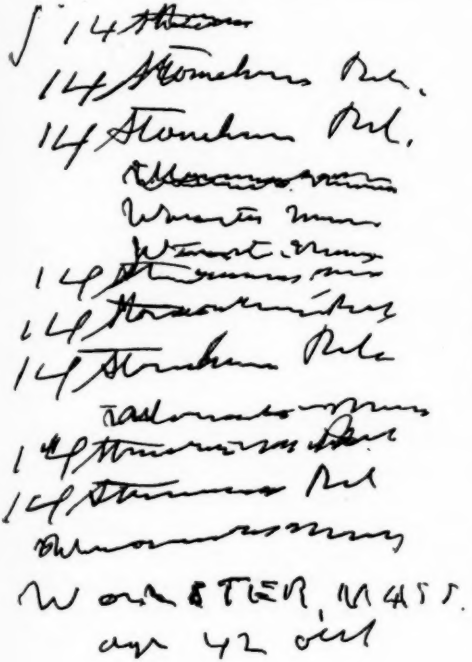

FIG. 1.

2. Write the letters of the alphabet.

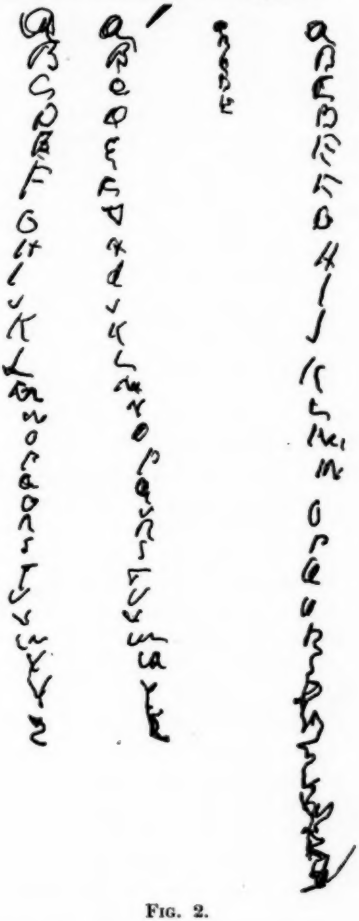

FIG. 2.

in the frequency with which the patient manifested speech perseveration. When it occurred it was usually of the nature of complete sentences rather than of single words. Sometimes he said the same thing over and over with somewhat different words, but the same idea was obviously repeated. By the end of two months the tremor had disappeared. The speech perseveration occurred only occasionally, as

after a long conversation or when the patient was fatigued or excited. On these occasions the tremor of his hands also recurred.

The use of the legs returned gradually and by the end of September, 1935, he was able to walk and run the length of the ward. He was extremely underactive and usually had to be escorted to meals and to the bathroom. Urinary incontinence sometimes occurred. His stance was generally one of flexion of the head on the trunk, the trunk on the pelvis, the arms at the elbow. It was noticed that he walked and ran on his toes, with his body leaning forward, and that his steps were short and rapid (propulsion gait). After a short demonstration of walking and running he appeared to have difficulty in keeping from falling. He would thrash his arms about and lean against a nearby wall or bed. When not within reach of some such support, he would fall to the floor, his knees slowly folding under him—so slowly in fact, that it was always possible for him to break his fall by catching himself on his hands.* At this time neurological examination revealed ironed-out facies, bilateral ankle clonus, and exaggeration of all deep reflexes. There was no muscular weakness or spasticity and no disturbance of deep or superficial sensibility. He still showed disorientation and profound memory impairment.

About four weeks later he left the hospital without permission. He was found a few hours later two miles away, having covered the distance on foot. Shortly after this incident he was discharged to his home. There was no change in his general physical condition; neurological examination was the same as at the preceding examination. He was disoriented and had marked memory disturbance. His speech was unusual only with respect to the occasional repetition of sentences and ideas when he became excited or fatigued.

DISCUSSION

In this case, as in many others reported in the literature, the psychiatric picture is probably complicated by factors other than those referable merely to carbon monoxide intoxication. In Wolff's case, for example, alcoholism, manic-depressive psychosis, and arteriosclerotic changes were pointed out as of possible significance. In our patient there is a family history of manic-depressive psychosis and congenital mutism. Furthermore, it is possible that the present picture is coloured, perhaps conditioned—at least to a certain extent—by the depression which preceded the exposure to carbon monoxide and which may still be present. In addition to these factors, one must also bear in mind the patient's previous personality 'make-up.' From available information it seems likely that he has been an impulsively weak, affectively poor, passive autistic, perhaps schizoid psychopath. What the significance of such complex psychopathic manifestations may be in a total picture in which intoxication is apparently in the foreground is difficult to evaluate; that they play some rôle is, however, more than likely.

There appears to be undoubted evidence of damage to the basal ganglia, particularly to the corpus striatum, as witnessed by the neurological signs of mask-like facies, general flexor stance, tremor (which has since disappeared),

* It may be of significance that, when the patient was asked why he fell, he once answered, 'I guess because I'm looking for sympathy.' Such 'insight' is probably more apparent than real since he may merely have been expressing comments which other patients had freely offered to him on his gait disturbance.

general poverty of movement, and also, probably, the propulsion gait. Cerebral damage is also to be inferred on the basis of the attentional defects, associative and memory disturbances, and dementia. The Parkinsonian picture may include manifestations which are contributed by a depressed stupor; at no time since coming out of coma, however, has the patient's mood been one of depression. Rather it has been one of apprehensiveness, apathy, or empty cheerfulness. There is no retardation, nor has it ever been observed; commands are executed in a rapid, though clumsy, manner. Because of the neurological signs which point to these regions of the brain, it seems permissible to regard the specific psychiatric disorders which interest us in terms of disturbed cerebral and, especially, basal ganglion function.

The features which we wish to emphasize in this case are the speech perseveration and the astasia-abasia. The rarity of occurrence of these symptoms in carbon monoxide poisoning has already been referred to. Merzbach,[3] on the basis of an extensive review of the literature and 24 personal cases, has ascribed pathological repetitive speech to the caudate nucleus of the basal ganglia, particularly the anterior portion. We feel justified in relating the speech perseveration to the corpus striatum because of its intimate clinical association with the tremor; the tremor was most pronounced when the speech perseveration for single units was most conspicuous; later in the clinical course they improved together; under excitement and fatigue both became more pronounced; and although the tremor disappeared before the cessation of speech perseveration, both returned on those occasions when excitement or fatigue supervened. One cannot, however, say that the speech perseveration is of the same nature as the tremor, indicating with such a statement that the mere repetition of words is nothing more than repetitive laryngeal movements similar to the manual movements of the tremor. Speech is obviously more complex than so simple a formulation can account for. Furthermore, the fact that writing disturbances were similar to those of speech takes the repetitive phenomena out of the realm of simple muscular (laryngeal) repetitive activity.

It is interesting, from a psychological point of view, that the recovery of speech was of the order of a progressive grouping of increasingly complex units. At first the patient repeated continually merely the last word of the examiner's question (repetitive echolalia); then he began to *answer* repetitively with only one or two words; as time went on, phrases of greater and greater length were spoken in a repetitive manner. His answers have always been relevant. At the present time (four months after the onset), repeated single words occasionally crop up in a sentence. Altogether, the general course of speech recovery may be said to have been in the direction of an increasing grouping of words, from single words to complete sentences. Whether or not the first transition, from repetitive echolalia to repetitive single words as answers, may also be considered of the same order of ' graded ' psychological function as the later increasingly complex word-grouping, is a

matter of speculation. One may also speculate upon the significance of the more complex configuration of the speech and writing expressions in terms of an increased capacity to group ideas as due to or in spite of the automatization of motor activity which has characterized the patient's clinical course.

The astasia-abasia, as another unusual feature of the clinical picture, must be regarded as a hysterical inclusion within the picture of organic brain damage (cf. Oppenheim). The patient's gait was propulsive in character; it seems uncertain whether it also is a hysterical manifestation. The hysterogenic nature of the astasia-abasia is indicated by the fact that in falling the patient always lowered himself carefully to the floor, that he seemed to utilize the fall for the sake of receiving sympathy, especially in the presence of an audience, and that on one occasion he walked for two miles with no difficulty. Wolff ascribed the astasia-abasia of his patient to the basal ganglia, indicating thereby the possibility of localization of hysterical manifestations in this region of the brain. We are not ready, on the basis of the clinical findings in our patient, to be in complete accord with this concept because of the complicating features, such as personality psychopathy, depression, and probable organic cerebral changes, in addition to the basal ganglion picture. On the other hand, the association of the astasia-abasia with the speech perseveration may argue for a common localization of these neuropsychiatric signs. The rôle of the corpus striatum, indeed, seems to us most significant in this connexion. Although it is granted that this region of the brain does not act independently of others, it seems plausible to ascribe the phenomena under consideration, at least in part, to its dysfunction.

Let us return to an analysis of the speech perseveration in our patient. There seems to be revealed that the repetition of digits, words, phrases, and sentences, perhaps of ideas also, may be quite independent of mere 'automatization.' Rather it may be due to a state of generally increased suggestibility. An action, a word, a group of words, an idea, may serve as strong suggestion to a repetition of that action, word, group of words, idea, for a more or less indefinite period. The tremor, by virtue of its intimate association with the speech perseveration, may also fall within this category of explanation, but we hesitate to speculate upon this beyond mere mention. The astasia-abasia, as a manifestation of hysterical psychopathy, can readily be accounted for on the basis of increased suggestibility. In the light of this general factor (hypersuggestibility) it seems possible to explain the origin of the speech perseveration and the astasia-abasia. These clinical manifestations, on the basis of such an explanation, become more than mere reactions of the nature of released primitive movements due to corpus striatum damage, but rather become understandable as expressions of a personality under certain changed physiological conditions. One may be permitted the generalization that suggestibility and its deviations in a plus or minus direction may be intimately associated with basal ganglion function. Such an observation is borne out not only in our case but also in that large group of postencephalitic

pictures in which changes of suggestibility are a prominent feature, particularly with respect to anancastic ideas and actions. The catatonic syndrome may also be referred to in this connexion. It is not to be inferred from these statements that we consider suggestibility to be localized in the basal ganglia. Rather we hypothesize that by virtue of the specific diminution or outfall of certain basal ganglion functions, due to organic damage, there is brought about a lowered threshold ('increased suggestibility') for the repetition of activities initiated from higher centres (the cerebrum). This hypothesis may also be stated in terms of disturbance of inhibition of corticofugal impulses by basal ganglion outfall. Our patient's diminished critical capacity, referable perhaps to cerebral changes, may also contribute to his hypersuggestibility.

We would like to suggest that the consideration of the neuropsychiatric significance of these manifestations in terms of changed suggestibility might also be applied to other clinical disorders in which basal ganglion dysfunction plays a prominent rôle. In any event, it appears to us that the speech perseveration and the astasia-abasia in this patient's clinical picture are most readily understandable by means of this formulation.

SUMMARY

A case history is presented of a patient in whom speech perseveration and astasia-abasia became manifest subsequent to carbon monoxide intoxication. Only one similar case (Wolff) has been reported previously in which both these signs occurred, and there is only one other brief mention (Oppenheim) of the occurrence of astasia-abasia following such intoxication. These clinical manifestations are ascribed to basal ganglion dysfunction. The hypothesis is formulated that, under such physiological conditions, there may occur changes of suggestibility by virtue of which this unusual picture may be described and explained.

REFERENCES

[1] WOLFF, G., 'A Case of Astasia-abasia and Speech Perseveration following Carbon Monoxide Poisoning,' *Jour. Neurol. and Psychopathol.*, 1926–27, **7**, 213.

[2] OPPENHEIM, H., *Lehrbuch der Nervenkrankheiten*, 1913, **2**, 1533.

[3] MERZBACH, A., 'Die Sprachiteration und ihre Lokalization bei Herderkrankungen des Gehirns,' *Jour. f. Psychol. u. Neurol.*, 1928, **36**, 210.

A CYCLOPS AND A SYNOTUS

By

K. H. BOUMAN, Amsterdam, and V. W. D. SCHENK, The Hague

INTRODUCTION

Only a small number of cases of cyclopia in human beings and mammals have been minutely examined. The number becomes still smaller if a more or less complete microscopic investigation of the central nervous system is stipulated. It is really only the cases of Davidson Black and Winkler and perhaps that of Naegli which answer this requirement. In contrast therewith there is an abundance of experimental studies in this field in urodela and other lower classes of animals.

For all that, unanimity does not by any means prevail here, although the views of Stockard and his followers—who held that the first determination of the eye lay unpaired in the median line—and those of Spemann—who pointed to a paired rudiment from the outset, which views were originally diametrically opposed—appear to have drawn somewhat nearer to each other in recent years.

Woerdeman, for instance, found that the paired rudiment of the eye shifts its position laterally downwards very early (when the folds of the medullary plate become visible) and he rightly says that this is not the same as Stockard's lateral growth of an unpaired eye rudiment. Yet, by saying this, he admits certain changes and growth conditions to which Fischel, for instance, did not do full justice. E. Manchot, on the other hand, who defends Stockard's views, admits that between the two regions of the eye rudiment there must be a tract of brain tissue (lamina terminalis and regio chiasmatica). She maintains that the regio chiasmatica belongs to the eye rudiment but admits that this does not imply that the last word about the determination itself has been said.

Petersen suggested three possibilities :—

(1) in the material of the presumptive medullary plate there are two separate groups of cells for the left eye and right eye ;

(2) the original unpaired rudiment divides into two ;

(3) the original unpaired rudiment grows sideways.

Woerdeman modified the second possibility slightly ; the original unpaired rudiment divides into three parts, viz. two eyes and the regio chiasmatica.

Of these three possibilities, the experimental researches of Spemann, Woerdeman and others have shown with certainty that the third is precluded.

If our general survey of literature is correct, no choice can yet be made between the first and second possibilities. Experimental embryology, therefore, has not yet succeeded in furnishing certainty on the point whether in the case of cyclopia one should speak of a defect or of an inhibition (Hemmung). For if it assumed that the eye rudiment is originally unpaired, it follows that pure cyclopia is a consequence of an inhibitory action ; but if a paired ' Anlage ' is assumed, one cannot avoid presuming that in the case of cyclopia a median sector has disappeared.

For that matter, put this way, the question has not been correctly formulated. It is inconceivable (at any rate if teratology is taken into account) that a defect should not at the same time have an inhibitory effect. That being so, the question becomes a different one and might be worded as follows : Are there any known cases of malformation in respect of which one can speak *solely* of inhibition ?

Our experience relates only to malformations of the nervous system and to the literature in this field. It shows that in the case of malformations of the central nervous system symptoms of inhibition frequently occur, but that there are no cases in which it has been conclusively proved that there are no defects. On the contrary, the general impression is that the same defects are nearly always found. However, *no* certainty in this sphere can be expected to be derived from teratology, which has to consider cases so much more complicated and composite than the physiology of development has to do.*

A teratologist cannot solve these problems. He is however in a position to describe his material as accurately as possible and to disclose valuable conditions and relationships in regard to certain functions of development.

Very special difficulties are encountered in examining cyclopia.

Much as is known about the formation of the eye out of the cerebral vesicle, little is known with certainty about the history of development of the telencephalon in the neighbourhood of lamina terminalis, infundibulum, and recessus opticus. Such knowledge is needed, however, to grasp the nature of the brains of cyclopes, and indeed to comprehend the formal genesis of cyclopia.

In this respect too, Woerdeman made an important research, whilst in regard to the end of the sulcus limitans at the neuroporus anterior there are numerous observations by neuroanatomists (Winkler, His, Kupfer, Bok).

In our opinion we are still far from having a clear insight into the history of development of these parts of the brain in higher classes of animals.

HUMAN CYCLOPS CASE

The first case of human cyclopia which is described here was discussed in 1909 by Prof. W. M. de Vries and by one of us (B.) at the Netherlands

* It is e.g. conceivable that changes in tissue which originally represent an inhibition may through secondary changes (resorption, fusions) present the appearance of a defect.

Physical and Medical Congress. Prof. de Vries stated at the time that in this case there was only one eye fissure which was bounded by four eyelids (this is still clearly visible in the photographs). The eye was perfectly simple. There was no trace of a nose. The philtrum was also lacking. In X-ray photographs and after preparation of the skeleton the following changes were found (fig. 1).

In the cranial bones there was a wedge-shaped defect situated medially, the acute angle of which passed through the sella turcica. That part of the os sphenoidale which usually lies between the two foramina optica was wanting. The foramen opticum was simple. Os ethmoidale, ossa nasalia, lacrymalia and a part of the os frontale were wanting. The remaining part of the os frontale was simple and small. The intermaxillary bone with the rudiment of the four incisors was absent. A medial part of the roof of the mouth cavity was absent from the inner surface of the skull, in consequence of which the vertical branches of the ossa palatina were very close to each

FIG. 1.—Skeleton of the cyclopian head.

other and the vomer was also wanting. The cavity of the nose and choanæ were entirely absent. X-ray films showed that on both sides of the lower jaw an incisor was wanting.

In regard to the condition of the perfectly simple eye Prof. de Vries stated at the time that it was not quite normal. Behind the eye there was a coloboma and a cyst. This cyst had a glia wall, an ependymal lining and was directly continuous with the optic nerve. Nervus opticus and cyst were enclosed by one connective-tissue sheath. This was therefore a cyst of the eyestalk. The cavity of the cyst was homologous with the cavity of the stalk of the primary eye vesicle. One of us (Bouman) then stated certain particulars regarding the general structure of the nervous system in this case.

EXAMINATION OF THE BRAIN

The following can be said of the examination of the brain.

It shows the typical structure of a cyclops' cerebrum. Medulla oblongata, pons Varolii and cerebellum are of normal structure.

When the cranium was being opened a bladder-like structure was seen to hide cerebrum and brainstem. On being incised, the ' bladder ' was found to communicate with the aqueduct of Sylvius, which opens crater-shaped into this cavity. When looking at this crater-shaped opening from above, one receives the impression that underneath it there are parts of the optic thalamus. An epiphysis and both habenulæ, which run frontal-wards, are visible. An apparently unpaired piece of brain tissue without furrows or convolution lies in front of this optic thalamus without any connection (figs. 2 and 3).

Seen from underneath, the infundibulum and an apparently unpaired optic nerve lie closely proximal to the pons.

The immediately adjacent broad piece of brain tissue seems to be fused here with the diencephalon. From underneath the distal parts of the telencephalon are seen to be paired and show a few furrows. The dorsal vascular membrane ' bladder ' arises

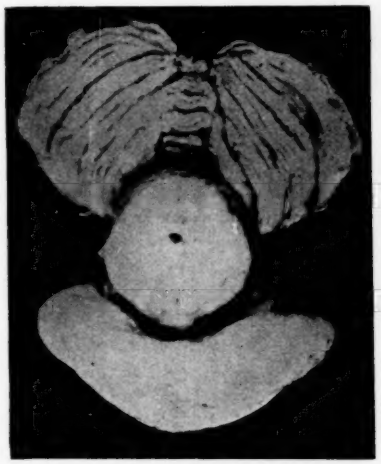

FIG. 2.—Cyclopian brain seen from above.

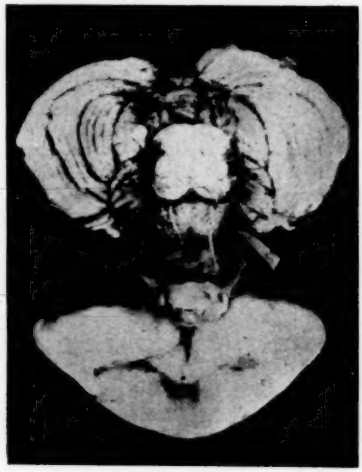

FIG. 3.—Cyclopian brain seen from below.

from epiphysis and habenulæ and disappears downwards between optic thalamus and prosencephalon.

The *microscopic examination* of this brain revealed the following (Schenk).

The *medulla oblongata* is seen in the sections to be well formed and larger than the normal control sections. The enlargement is almost entirely in the olives (fig. 4).

On measuring the surfaces the following figures were found :

	Normal.	Cyclops.
Olives	100	135
Tegmentum	100	106
Med. oblong. tot...	100	117

These figures have been reduced to 100 for normal. The place for fibres of the pyramidal tract has not been included.

Olives and inferior olivary bodies are well developed. Pyramidal tracts are entirely wanting. The lemnisci mediales exist, but are little developed. All other nuclei are normally represented together with their roots, primary and secondary fibres. The maturation of the medullary sheath appears to have advanced further than in

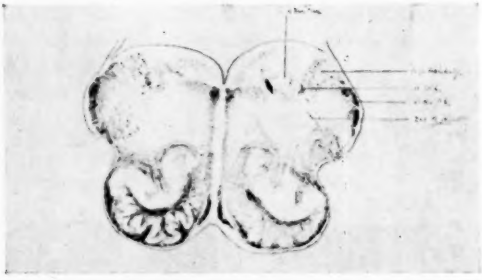

FIG. 4.—Drawing of a section through the medulla oblongata. The olivæ inferiores are seen here at the place where they are most enlarged.

normal preparations of a neonatus. When examining the nervous system further in a proximal direction, one meets with anatomical pictures which differ very little from those of a normal child. We shall hereinafter not discuss the points of resemblance with normal brains. Nervi abducentes, faciales and octavi, with their nuclei

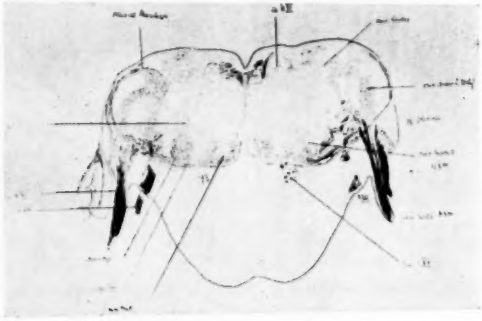

FIG. 5.—Drawing of a section through the pons. On the left the nervi vestibularis and facialis are visible ; on the right both parts of the nervus octavus.

and secondary fibre systems, are undamaged. The pontine nuclei, too, exist and are normal (fig. 5).

The cerebellum is fairly large. The lamina granularis externa is still entirely present. The maturation of the medullary sheath has advanced further than in normal preparations (the neocerebellar parts are also well myelinated). Deviations were found in the nucleus dentatus. Instead of one cell-band, numerous separate groups of cells are found side by side. Other windings are very small. This is therefore microgyria and fragmentation.

The nervus trigeminus with its spinal and mesencephalic tracts and nuclei is of a normal structure.

Just above the pons the mesencephalon assumes an unusual appearance. At the decussations of Meynert and Forel the infundibulum with both corpora mammillaria is visible ventrally in the section. The pedes pedunculorum are entirely devoid of fibres. They are crossed only by the roots of the nervus oculomotorius. As far as is ascertainable, the nuclei of this nerve are normal. The nervus trochlearis, too, has no deviations of importance.

In these sections the red nucleus becomes visible very soon after the disappearance of the pons. The dorsoventral size of the sections is large here. Ventrally, in the median line, there are numerous incoming fibres which cross and recross in an irregular manner. Dorsal to the nervus opticus lie the two corpora mammillaria and between them lies the infundibulum. Lateral to these there are only a few fibres and

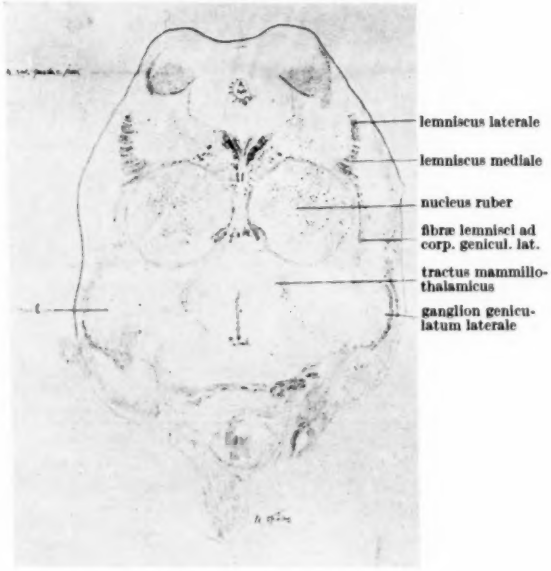

FIG. 6.—Drawing of a section through the mesencephalon.

cells. The root fibres of the nervus oculomotorius intersect the red nucleus. Laterally and dorsally to this lie the two lemnisci, which lose fibres in a dorsal direction. The trochlearis roots cross dorsally to the aquæductus Sylvii.

The red nucleus reaches its greatest diameter in the next sections. The surface is considerably greater than in normal preparations (fig. 6).

The nervi optici do not form a distinct chiasma although a few fibres cross. On both sides these fibres are gathered into a fairly well-defined bundle. A part of them end in a nucleus, which also derives fibres from the lemnisci: ganglion geniculatum laterale. Laterally to the infundibulum lie two little bundles. One of them is fairly well medullated and the other slightly. The bundle which is deficient in myelin disappears proximally in the nuclei thalami and is possibly the mammillothalamic tract of Vicq d'Azyr. The other one soon curves lateroventrally and disappears in the mass of fibre which fills the ventral parts of the section.

In the next sections the radiatio optica can be followed further. After passing the ganglion geniculatum externum, in which a number of fibres terminate, other optic fibres radiate in the corpora quadrigemina which are formed in the dorsal parts of the mesencephalon. In this section the red nucleus loses, in a ventrolateral direction, fibres which disappear proximally in the nuclei thalami. The lemnisci also split up into fibres which run ventrally.

In the sections which succeed these, the red nucleus ends in a ventrolaterally directed radiation. The lemnisci, too, gradually merge. The radiatio optica also produces the lateral and ventral limits of the section. The fasciculus retroflexus of Meynert now occupies, as it were, the place of the red nucleus. Ventrally the first parts of the thalamus appear. In this section (fig. 7) it is probably the ventral nucleus

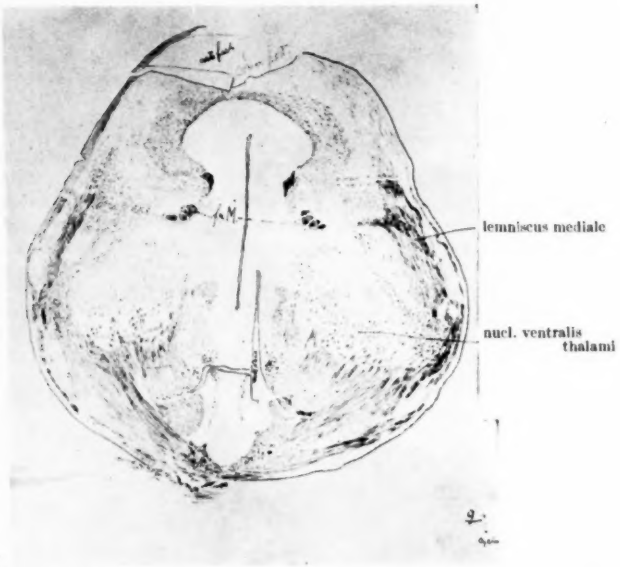

FIG. 7.—Drawing of a section through the proximal part of the mesencephalon. The nucleus ruber merges here in the thalamus.

which shows a broad radiation ventromedially. Recognition of the various nuclei thalami presents great difficulties, and for this reason a few names have, hereinafter, been applied with some degree of hesitation; the anatomical situation is aberrant; the capsula interna is wanting, and the corpus striatum likewise.

In the following section the corpus quadrigeminum anterius and the commissura posterior are visible. Laterally to the fasciculus of Meynert lie complexes of nuclei, which show some resemblance to the nuclei præbigeminales. There are, ventrally to them, a lateral nucleus which has derived fibres from the nucleus ruber, a nucleus ventralis and a nucleus reticularis, lying in the radiatio thalami. The latter forms a medial crossing.

In the next section the radiatio thalami turns towards the hemispheres which here lie ventrally to the thalamus. The thalamus has only a few fibres with myelin. It is

not certain whether there is a nucleus subthalamicus among these centres, which are but little distinguished ; there is no trace of a corpus striatum.

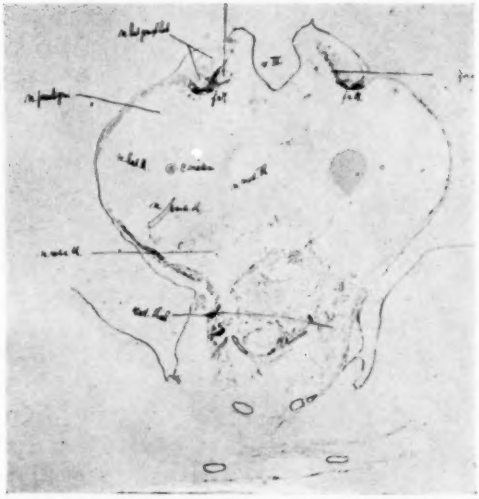

FIG. 8.—Drawing of a section through the diencephalon after opening of the aquæductus Sylvii in the third ventricle. Ventrally telencephalon and diencephalon are fused.

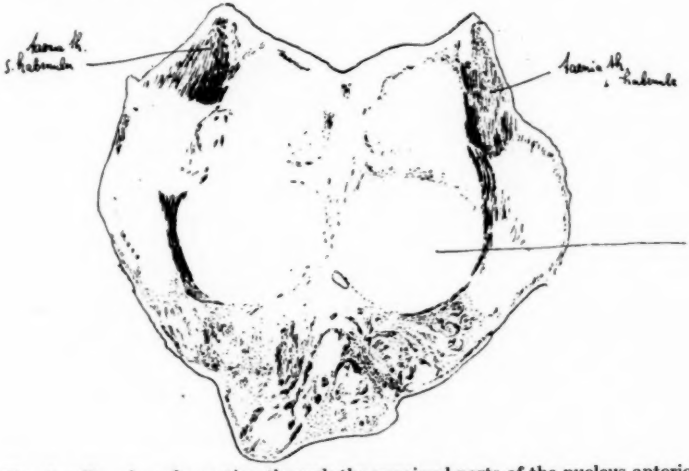

FIG. 9.—Drawing of a section through the proximal parts of the nucleus anterior thalami in the diencephalon.

In the section which now follows there are a few fragments of the epiphysis. A number of cells are grouped round the fasciculus retroflexus, whilst a few fibres proceed dorsally towards the middle line (pes conarius).

In the next section (fig. 8) the aquæductus Sylvii opens up. There is on both sides a finely developed ganglion habenulæ with a nucleus lateralis and medialis. Not much change takes place in the mass of the thalamus. Ventrally, a broad irregular mass of fibres forms a connection with the hemispheres (radiatio thalami).

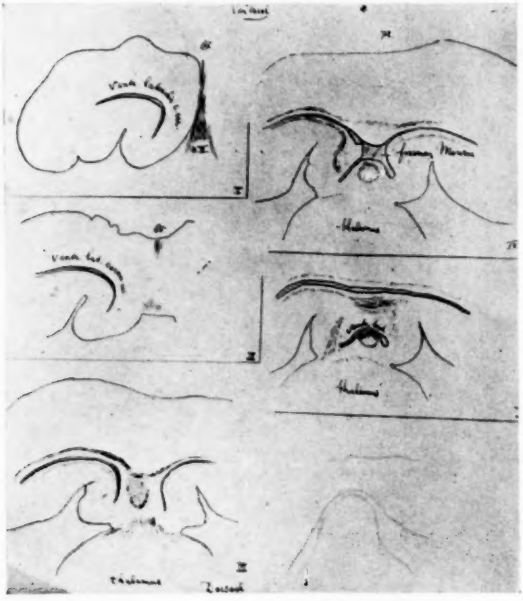

FIG. 10.—Diagram of ventricles in the cyclopian brain.

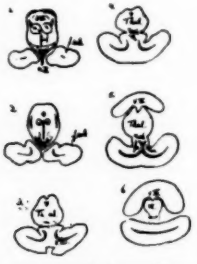

FIG. 11.—General aspect of mesencephalon, diencephalon, telencephalon and ventricles in the cyclopian brain.

In the next section the ganglion habenulæ has merged into the tænia thalami. The thalamus has diminished in size. In the sections which now follow, the size of the diencephalon rapidly diminishes. A broad bundle emerges from the tænia thalami, curves ventrally and disappears into the broad ventral mass of fibres. The nuclei thalami merge here into a single round nucleus (nucleus anterior) (fig. 9).

Telencephalon.—The telencephalon lies entirely ventrally to the diencephalon. Seen sidewise it is flat (dorsoventral dimension); seen from above it is round and shaped somewhat like a horseshoe in the occipitally situated part. The relationship between the ventricles has been made clearer in the diagrams (figs. 10 and 11). After changing into the third ventricle the ependyma of the aquæductus Sylvii covers the dorsal surface of the thalamus between habenulæ and tæniæ thalami as far as its front end. Then returning in a backward direction, the ependyma can be followed in a wide funnel lying between thalamus and forebrain. Finally, this canal ramifies in three directions in the brain itself. An ependymal fissure enters each of the two occipital lobes, whilst one branch enters the forebrain. The place where this division occurs might rightly be called the foramen of Monro. The third ventricle which dorsally forms a large 'bladder' (this has not been preserved in the sections, but is distinctly shown in the photographs of the undivided preparation) therefore narrows

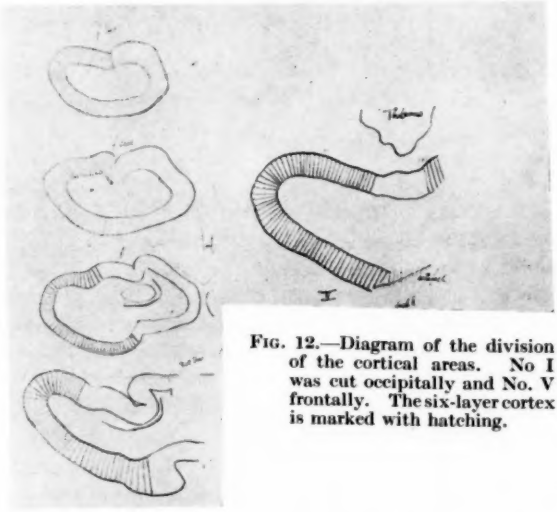

FIG. 12.—Diagram of the division of the cortical areas. No I was cut occipitally and No. V frontally. The six-layer cortex is marked with hatching.

ventrally to a funnel, which in the case of a foramen Monroi divides into side ventricles. The cortex of the great brain is not built in the same way everywhere. In a few places there is a cortex consisting of six layers, such as one would expect to find in the neocortex of a newborn child (indicated in the diagram by hatching). Dorsally as well as ventrally the cortex is built differently in the median line. It is not difficult to distinguish in it a hippocampus, parts of a nucleus amygdalæ and olfactory cortex with verrucæ Arnoldi (left white in the diagram) (fig. 12).

In order to elucidate the complicated anatomical situation, we will describe a section near the foramen Monroi. The shape of the whole resembles somewhat that of an anchor, whilst the cavity of the ventricles looks much like an inverted V. The wall of the cerebral cavities has collapsed entirely. Basally, there is a strip of cortex with verrucæ Arnoldi; to the left and right it is continuous with the neocortex which occupies the lateral parts wholly. Not until it comes to the neighbourhood of the median line does this neocortex change dorsally into another type: the hippocampus formation. Without a distinct fornix a fusion takes place with the thalamus

here in the mediodorsal line. The fibres connecting cortex and thalamus run through this fusion. They form two groups : one group of fibres comes from the distally situated parts of the cortex and is connected with proximally situated parts of the thalamus. Another group springs from the more proximally situated parts of the cortex and goes at practically the same level in a left and a right bundle towards the thalamus. Both groups cross each other sagittally, so that the distal fibres end in the thalamus proximally to the other cortex fibres. There is also a crossing of fibres from the right and left hemispheres. This also applies to the corticothalamic fibres. These also cross in the median line.

In the spinal cord there are no great deviations except the absence of the pyramidal tracts. It gives the impression of being small.

SUMMARY

Recapitulation of the deviations in the central nervous system :—

A series of general disturbances in those parts of the brain which are not directly linked up with the eyes or olfactory brain : anomalies in the nucleus dentatus, changes in the relative size of olives, red nuclei and the other parts of the myelencephalon and mesencephalon. Absence of pyramidal tracts and corpus striatum.

A number of disturbances in the proximal parts of the nervous system :—

1. Absence of the chiasma opticum, but existence of a tractus opticus which on both sides goes to the homolateral ganglion geniculatum externum.
2. Absence of the hypophysis cerebri, pars posterior.
3. Absence of bulbus and tractus olfactorius.
4. Absence of fornix and corpus callosum.
5. The third ventricle has become a large ' bladder ' dorsally.
6. The foramen Monroi has been displaced ventrodistally and is under the thalamus.
7. The medioventral parts of the diencephalon (corpus mammillare, infundibulum and tractus opticus) have been displaced distally.
8. The lateral parts of the diencephalon have been displaced ventromedially (radiatio thalami) and a decussatio thalami has arisen.

CALF SYNOTUS CASE

Prof. C. Winkler kindly enabled us to study a case belonging to his collection, and placed at our disposal a series of sections of the central nervous system and a few photographs of a misshapen calf.

The calf in question, when examined with the naked eye, showed a large number of severe malformations. The photograph (fig. 13) shows the front of the head with two ears, which are near each other in the median line. Nothing can be seen of eyes or of a nose. In the middle there is a hole, about which we possess no information.

It is possible to conclude from X-ray films that the occipital and parietal bones are well developed, that there are two large petrosal bones and that the facial skull has been reduced to a meagre residue which it is difficult to unravel.

In the photograph of the brain preparation (fig. 14), as removed from the skull, it is possible with the unaided eye to discern the following remarkable situation.

Whilst the medulla oblongata and the cerebellum are normal in appear-

FIG. 13.—Front aspect of the synotical calf.

ance, the proximal parts of the brain are completely abnormal in structure and outward appearance. When examined with the unaided eye, this part of the brain reminds one of the earliest stages of development of the nervous

FIG. 14.—Brain of the synotical calf.

system, when the thalamus is high up cranially and the telencephalon curves sharply ventrally. But here at this bend the comparison ends, as there is no trace of eye vesicles or windings in the ventrally curved proximal piece. This mass does, however, show a fairly sharp sagittal fissure.

In the prolongation of the metencephalon and mesencephalon lies a

membranous vesicle which, compared with the 'bladders' that are usually found in cyclopes, may be said to be very small. When this vesicle is opened, the crater-shaped opening of the third ventricle is visible (as will be seen later on). Many tela chorioidea are found here.

MICROSCOPIC EXAMINATION (SCHENK)

The preparations had been previously put into chromate and stained alternatively, according to the Weigert-Pal method, with hæmatoxylin and carmine.

The nuclear systems and fibre systems of the medulla oblongata, of pons and cerebellum only show a few deviations. Whilst the nuclear and fibre systems of the nervi XI, X, IX and VIII are strongly developed, the nervi XII, VII, VI and V are represented only by a few fibres and nuclei. However, none of these nerves has disappeared or been reduced beyond recognition. The cerebellum is also completely developed. The fibres are well medullated and the cortex is normally built. In the mesencephalon the posterior corpora quadrigemina are strongly developed. The place of the olivæ superiores is indicated on both sides by a dense network of fibres, but they are extremely deficient in cells.* The nuclei trapezoides mediales have large cells. The nuclei trapezoides laterales have disappeared entirely. In the corpus quadrigeminum posticum the brachium posticum, the connecting arm with the corpus geniculatum mediale and the cerebral cortex, contains very few fibres. The cells in the corpus quadrigeminum posterior are small and few in number. The corpora quadrigemina anteriora are fairly strongly developed. In them various layers are found, which decussate in the case of mammals, and many cells of normal size, with the exception of the large cells of stratum griseum intermedium, which are wanting.

When continuing the series in a proximal direction, fibres and cells of the nervus oculomotorius are found. They are weak, but distinctly developed. Medioventrally lies a large ganglion interpedunculare. The part with large cells of the nucleus ruber is large and well developed. In the mediodorsal parts of the corpora quadrigemina there are numerous transversely cut fibres which are gathered in a bundle. This bundle derives fibres from the layers of fibre of the corpora quadrigemina and increases more and more in size in a proximal direction.

In the sections which follow now, the structure of the corpora quadrigemina anteriora disappears. The dorsomedian bundle remains. Between the commissura posterior and aquæductus Sylvii one can see the distally situated parts of the subcommissural ependymal organ, which is of large extent. Remains of eye muscle centres lie between the fasciculi longitudinales posteriores. It is possible to distinguish a lemniscus medialis and lateralis. The latter continues to lie laterally and it ends, in the sections which now come, in a centre, which is therefore in all probability the corpus geniculatum mediale. The first (lemniscus medialis) gradually begins to radiate in dorsolateral direction. Here is the beginning of the tract of Meynert with large ganglia interpeduncularia.

The changes which can be ascertained when proceeding further in a proximal direction are of two kinds. In the ventral parts of the mesencephalon an irregular connexion arises between the two halves which are prolonged on both sides laterally in tangential fibres. Not a single fibre of the nervus opticus can be traced. These

* This atrophy corresponds fully to the function of the nuclei olivares superiores and the nuclei trapezoides laterales connected therewith, as described by Winkler (Vol. II, p. 256).

ventral fibres form a decussation in the middle ; bundles which have been cut transversely can also be found in it, but it is not possible to trace any resemblance here to the tuber cinereum which should gradually appear here. The other peculiarity of these sections is the formation of the corpora geniculata. It follows from the situation and supply of fibres that the ventrally situated centre must be the medial corpus geniculatum, and the dorsally situated centre the lateral corpus geniculatum.

The formation of the epiphysis is visible in the sections which follow. Fully distally, a small quantity of gland tissue is cut, in which it is soon possible to ascertain a lumen. This opening rapidly increases in size. The gland tissue continues to exist in the dorsal wall of the tube for a considerable distance. Fibres then become visible, ventrally at first and also dorsally further on, and the gland gradually disappears. When it has disappeared the space is lined with a palisade epithelium. Still further

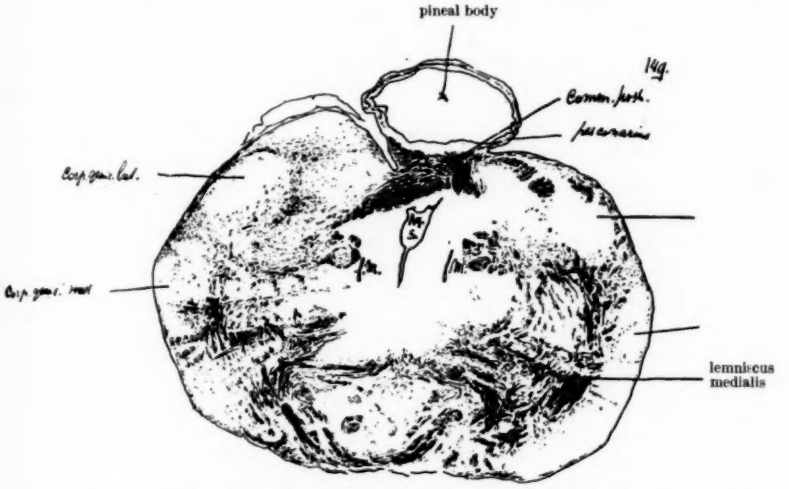

FIG. 13.—Drawing of the diencephalon of the synotus. There is no gland tissue here in the wall of the pineal body.

proximally the fibres in the wall of this epiphyseal organ are continuous with pedes conarii which disappear entirely in the corpora geniculata lateralia, in the commissura posterior, and are probably also connected with the dorsomedial bundles from the corpora quadrigemina anteriora (as far as they have not already disappeared in the corpora geniculata lateralia) (fig. 15).

In the ventral parts of these sections there is no trace of incoming fibres and no trace of a mammillary body. Perhaps one of the little bundles which has been cut transversely is a very atrophic mammillothalamic tract.

In the next section (fig. 16) aquæductus Sylvii and the cavity of the epiphyseal organ meet and form the third ventricle. The pedes conarii are continuous with the corpora geniculata. The fasciculi retroflexi approach the floor of the third ventricle. Here the lemnisci already merge partly in the beginning of the thalamus. Ventrally a connection has arisen between the telencephalon and the diencephalon. The fornix lies on both sides ventrally against the diencephalon.

In the next section the fasciculus retroflexus merges in the ganglion habenulæ.

The third ventricle does not increase in size. The corpora geniculata become smaller and increasingly poor in fibres. At the side of a radiatio thalami a nucleus anterior can be discerned. The fornix presses up to the radiatio thalami.

In the next section both ganglia habenularum can be seen to be well developed. Here the tænia thalami which arises from this centre already radiates partly into the nucleus anterior. The ventral parts of the section continue to be indistinct; the end of the fornix fibres cannot be determined. It is uncertain whether there are any nuclei here.

In the next section the size of the nervous system diminishes. Here the whole of the ganglia habenularum is continuous with the tænia, while the latter loses many

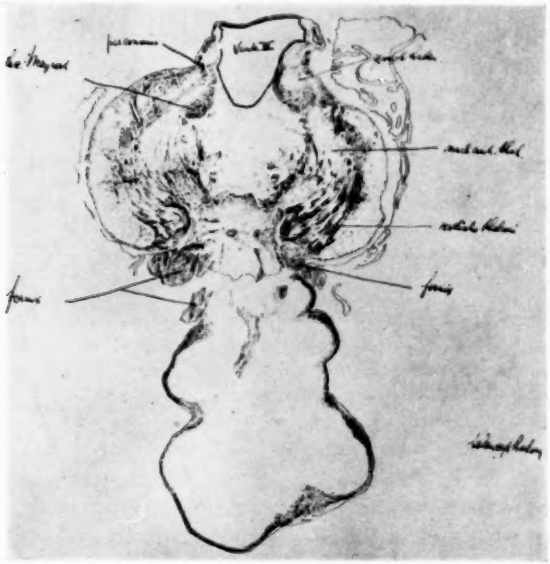

FIG. 16.—Drawing of a section through diencephalon after the opening of the aquæductus Sylvii in the third ventricle. Ventrally telencephalon and diencephalon are fused.

fibres in the nucleus anterior. In the dorsal roof of the third ventricle the plexus chorioideus is inserted here; the ventricle continues to be small and is continuous sidewise with a sort of side ventricle which is situated entirely lateroventrally. The foramen Monroi (?) therefore lies laterally to the thalamus here.

In the succeeding sections the nervous system ends quickly. In fig. 17 six sections through the telencephalon have been drawn. There is no trace of a ventricle. In the first section only the dorsal part is lined with ependyma, which is found to be the floor of a paired (?) ependymal 'bladder.' In the next sections, which are situated more proximally, this 'bladder' divides into two and a part of the lateral surface of the telencephalon is also covered with ependyma. When, proximally, the telencephalon again ceases to be connected with the thalamus, we find an unpaired ependymal 'bladder.' We receive the impression that in a few places a slip or funnel of the

ependyma penetrates into the telencephalon and cannot be followed any further in a vascular mass. It would have to be followed in distal sections medially to the fornices. It is not possible to decide whether the ependymal lining of the brain is primary or secondary : the so-called side ventricle might possibly have been fused subsequently with the brain in the former case ; in the latter case no ependymal funnel would penetrate the brain, and that part of the brain which is lined with ependyma would correspond to the cornu ammonis formation which is turned towards the ventricle. The solution of this problem presents great difficulties but is of extreme importance to the solution of the question how this nervous system is built. Unfortunately no good starting-point in the shape of ganglion cells can be found ; they are built irregularly. Nor are there any remnants of ependyma in the central mass, and if there had been any they would have been displaced by a vascular mass. The

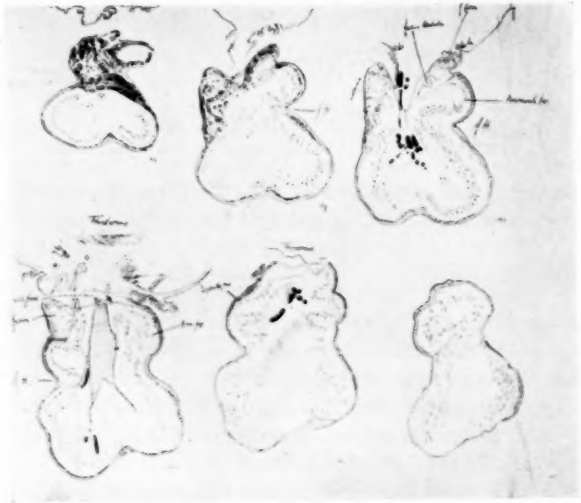

FIG. 17.—Six drawings of sections through the telencephalon of the synotus.

whole surface of the telencephalon is covered with a thin tangential layer of fibres. Dorsally this is continuous with the fornix. There are few or no fibres in the centre. At those places where the ependyma covers the surface, we may therefore think of the possibility of an alveus formation. In that case the tangential fibrous coat would have to correspond to the white matter, and this would force us to suppose that the brain vesicles were turned completely inside out—the white matter outside and the surface of the cortex inside. It is difficult to defend this supposition.

We should advance a step if in the ventrally situated parts there were parts of cortex with a distinct structure. To a certain extent this is the case. At a few places, immediately beneath the tangential layer of fibres, there is indeed a layer of cells which shows some resemblance to a second cortical layer. Underneath this lies a layer of ganglion cells which is broad and deficient in cells and corresponds to the lamina ganglionaris. This induces us to assume that the exterior layers of this brain are indeed the outer ones ; that there must be a collapsed ventricular cavity in the centre and that in this cortex the tangential fibres have continued to be the principal centri-

petal and centrifugal way of impulses, whilst the way along the central marrow has not developed at all (fig. 18).*

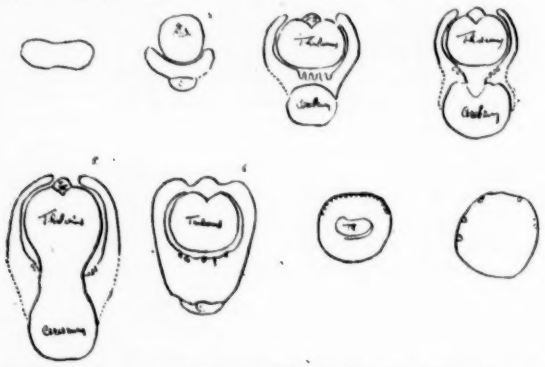

FIG. 18.—The proportions of thalamus, telencephalon and ventricular system in a synotus ; No. 1 has been cut distally, No. 8 proximally.

The foregoing has not solved all the difficulties, because the situation here is very complex and can in point of fact only be understood in connexion with other cases of cyclopia.

SUMMARY

Recapitulation of the deviations in the case of synotia in a calf :—

In addition to the profound changes of the facial bones, where the eyes were entirely absent and the nose and mouth cavities formed a single opening, the following abnormalities were found in the central nervous system :—

1. Absence of the large ascending and descending tracts running through pons, medulla and spinal cord (pyramidal tract, lemniscus).

2. Absence of nervus, chiasma and tractus opticus.

3. Absence of nervus, tractus olfactorius and all primary and secondary olfactory fibres.

4. Absence of the greater part of optic thalamus, corpus striatum, of corpus mammillare and hypothalamus.

5. Radical defects of the remaining parts of the telencephalon which, as in the previous case, have been displaced ventrally and reduced to a very simple form.

6. Formation of an epiphyseal organ with fibres derived, inter alia, from the corpora quadrigemina anteriora and geniculata lateralia.

DISCUSSION

When these two cases are compared with the cases described in the literature, a few points of similarity will be observed, besides many differences.

* This agrees with Davidson Black's findings in his case of cyclopia. In it, too, the tangential layer of fibres *alone* had developed.

Before proceeding to make comparisons we are justified in inquiring whether we have the right to bring together certain malformations of the proximal basal plate of the central nervous system.

Do the cyclocephali (Geoffroy St. Hilaire) and the synoti belong together and do certain forms of anencephaly also belong to this group ?

W. M. de Vries discussed in 1912 the grouping of these monstrosities. In agreement with Kundrat he calls the mildest deviations in this group *cebocephali* : in these cases the eyes are separated although the two orbits are close together, and the nose is still present rudimentarily. In the case of *ethmocephali* the separated eyes also lie near each other, but the rudimentary nose has taken the shape of a proboscis. In the case of *cyclopes* proper, both eyes are found to be more or less fused in a single orbit, and in addition various deviations of nose, mouth, ears, etc., occur.

G. St. Hilaire mentions cases in which in addition to deviations of the eye there is only a proboscis where the nose should be (rhinocephalus) ; cases in which the nose is only atrophic (cyclocephalus) ; cases where in addition to a proboscis an atrophy of the mouth can be found (stomocephalus).

Still more pronounced deviations link up in a natural manner with the foregoing. Where eye, nose and mouth cavity are united, the ears approach nearer to each other ; the defect in the median line is more extensive, the eye disappears, the ears approach nearer to each other, the nose disappears.

We therefore see a series ranging by gradual transitions from slight cebocephaly to the extremely marked anomalies of the synotus. Moreover, experimental embryology justifies the assembling of these monstrosities which are apparently so heterogeneous, because—apart from other deviations which are found in each of these monsters—there is in all, in common, a defect (possibly an inhibitory process) in the front wall of the neural plate in its earliest periods of development.

Former investigations rightly paid great attention to the slight cases, because these are so near the normal type. However, the severe cases also throw a clear light on these monstrosities and their formal genesis, because in them the disease has persisted radically. An example of this is given in each of the cases described.

They are therefore forms of monstrosities in which through some cause or other those parts of the embryo which lie in the median line basally to the neuroporus anterior are injured and remain retarded in development. In both cases extensive defects have been found, relating to the skin, organs of sense, bony cranium and brain.

A few points require further discussion : (1) The anatomy of the roof of the third ventricle and the place of the foramen Monroi and the telencephalon ; (2) the epiphyseal organ in the case of synotia ; (3) the relative size of nucleus ruber and olives in the case of cyclopia.

Winkler drew attention to the dorsal ' bladder ' which is said to be found on the plate of the third ventricle in all cyclopes. In substantiation

of this view he cites researches by Davidson Black and refers to his own experiences of cyclopes and to a number of cases mentioned in the literature. He considers that the ' bladder ' is responsible for this and other malformations and imagines that the primary hydropsy of the third ventricle (he left undiscussed the question how it arose) bursts ventromedially and destroys those parts of the embryo which are situated medioventrally to the neuroporus anterior. The fusion of eyes and forebrain is therefore alleged to take place secondarily and not primarily. It is in this manner that Winkler thinks it possible to avoid assuming a primary cause of inhibition of development.

The difficulty and danger of all teratological researches is the fact that one has to deal with scars and remnants of diseases and disturbances in the development of the embryo, which arose and terminated months previously in very young tissue, the regenerative power of which is completely, or at any rate largely, unknown to us. Consequently, the way is opened for numerous suppositions which cannot be proved.

Winkler's theory has not escaped entirely from these risks. He had however an apparently solid basis in the frequency of the hydrops ventriculi, which is also present in our cyclops. It was therefore justifiable to recognize in this ' bladder ' an important factor in the pathogenesis of these monstrosities. As opposed to the somewhat vague and unsubstantiated suppositions of Stockard's school, which still clung to a mysterious primary inhibition of growth, this view, which indicated a well-defined defect, was no doubt attractive.

However, the study of our second case, synotia, presents unforeseen difficulties. In order to obtain a general view of these data, it is necessary, however, to recapitulate what we found in the telencephalon.

If we wish to form a clear idea of the structure of the telencephalon in these two cases, we should recall to mind that the great brain also arises from a plate. The infundibular region therefore corresponds to the floor plate ; the plexus chorioideus which is attached to the fimbria fornicis corresponds to the roof plate ; whilst the brain cortex itself corresponds to wing and floor plate. (It is noteworthy that in the case of acrania, described by one of us, these original conditions were demonstrated in a fœtus of five months.)

In the accompanying diagram (fig. 19) the original situation is shown (A) and it is indicated what situation arises in a frontal section if the plate closes up and forms a tube (B). In both cases of cyclopia the walls of the tube collapsed in the end (C). In the case in which the brain cortex has developed more strongly, both side ventricles arise. In the case of synotia, the brain cortex continues to be very meagre. In the frontal section (fig. 20) the defect situated ventrally in the median line has still to be discussed. In both cases olfactory cortex is found medioventrally in the parts of the great brain. This is clear particularly in the first case. We are obliged to assume that we are dealing here with derivatives of parts of the cortex which are situated

medially in the front brain wall. As a matter of fact, the rhinencephalon is found in the diagrams of His proximoventrally to the pallium and proximodorsally to the primary eyestalks. There is however a defect in the median line. Here is wanting the margo reuniens (His) and what it changes into. Furthermore, the rhinencephalon has not developed, although there are pieces of cortex which have retained the structure (fig. 21).

In continuation of the foregoing, it is necessary, in the second place, to form an idea of the situation of the telencephalon in respect of the thalamus. This can best be done in sagittal sections.

Fig. 22 is a schematic drawing of such sections of our cases and of a few

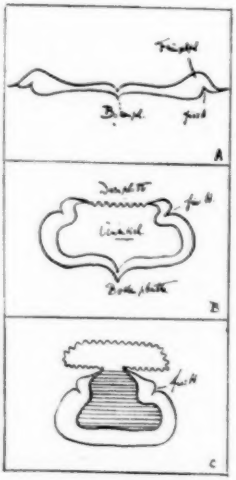

FIG. 19.—Diagram of tube-forming in telencephalon.

FIG. 20.—Diagram of frontal sections through four cyclopian brains.
W. : Winkler's cyclops.
D.B. : Davidson Black's case.

cases known to us from the literature. These drawings show that in our first case the telencephalon had been displaced ventrally and slightly distally to the thalamus. The connexion between the third ventricle and the side ventricles when these are formed after the division of the foramina Monroi consists of a tube which runs in a distal direction under the thalamus (the thalami optici are closely fused in the median line) and connects the third ventricle (inclusive of the dorsal ' bladder ') with the collapsed cavities in the telencephalon. Foramen Monroi is the name which we gave to the place where this tube divides into two. The back wall of the tube corresponds to the infundibular region, the front wall to lamina terminalis and roof of the front part of the diencephalon. The structure and relative size of the ventricular system of the synotus show a resemblance here to the foregoing in many respects, but they also show certain peculiarities. In the first place

the roof of the third ventricle is distended either a little or not at all. In the second place the entrance to the ventricular system of the telencephalon has collapsed entirely. In the third place, the connecting tube between the ventricles of the telencephalon and the third ventricle has collapsed for a certain distance in the median line and has remained open laterally only, so that there are apparently two side ventricles with a foramen Monroi here too. (A closer examination shows that this division into two only appears to exist.) For the rest, the same ventrodistal displacement of the telencephalon in respect of the thalamus is found here. How did this situation arise ?

Winkler assumed that through some cause or other a hydropsy of the third ventricle had arisen with a distension of the roof, with secondary breach and destruction of parts of the brain. He held this to be responsible for the

FIG. 21.—Diagram of the enlargement of the older parts of the cortex (dotted).

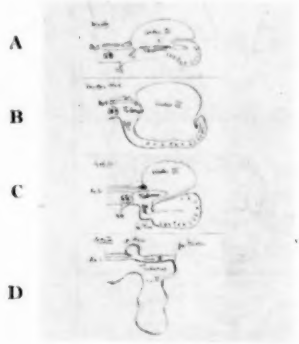

FIG. 22.—Sagittal diagrams of different cyclopes. A. The case of cyclopia described by Winkler. B. By Davidson Black. C. The cyclops. D. The synotus.

overturning of that part of the occiput in which the fissura calcarina is situated dorsally and not medially. The difficulty is, that in the case of synotia there is a ' bladder,' but that it is far from certain whether it should be considered to belong to the third ventricle. Furthermore, this hypothesis obliges us to make a series of suppositions which are rather uncertain (the fissure in the ventral parts, the regular defects which in many instances are found in the case of severe mechanical injury, the question why the hydropsy in this early stage has such serious consequences).

In order to acquire a good insight into the matter it is necessary to examine more closely the structure of the cortex of the brain of cyclopes. Winkler and Davidson Black drew a wedge on the surfaces which are seen basally, by which they effected an imaginary removal of a medioventral part of the olfactory convolutions. The remaining part showed much resemblance to the brains of their cases. This argument applies most forcibly to the

olfactory bulbs themselves, which are absent in every case of arhinencephaly. For the other parts of the brain, however, the situation is not so plain. In our second case little could be said with certainty about the matter, although there seemed to be a great similarity to the first case. It was possible to examine with considerable accuracy the remnants of the brain cortex of the human cyclops. In the drawings and diagrams given here, the structure of the cortex is indicated. These investigations showed (1) that the brains of cyclopes are not built unpaired, but consist definitely of two symmetrical halves (confirmation of older investigations); (2) that the olfactory cortex, notwithstanding the disappearance of the olfactory bulb, still occupies a fairly large surface of the medially situated parts of the brain (fig. 21).

We therefore find that important parts are wanting, but we also find

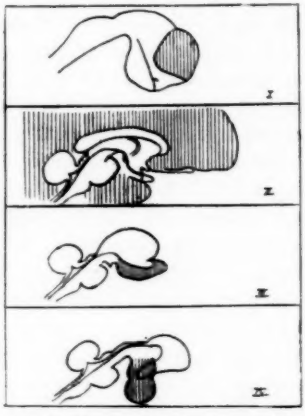

FIG. 23.—Diagram of origin of cylopian brain. I. Normal young embryonal brain. II. Normal adult brain. III, IV. Cyclopian brains.

that the telencephalon forms, in its shape and cytoarchitectonic structure, a primitive organ which has remained at a lower stage.

Both defect and inhibition can be clearly shown. How must we coordinate all these apparently disconnected data? They consist of (1) defects of the medioventral parts of the diencephalon; (2) membranous degeneration of certain medioventral defective parts and of the border planes between thalamus and telencephalon; (3) ventrodistal situation of the telencephalon in respect of the thalamus opticus; (4) inhibition of development as well as defect in the cortex of the telencephalon.

One of the possibilities which we wish to deal with, and in respect of which conjectures are utilized as little as possible as aids, is the following.

The situation of the telencephalon in respect of diencephalon and mesencephalon in both cases which have been examined here, recalls to mind the models of young embryonal brains known from the literature (figs. 23

and 14). If one supposes that the parts which lie distally to the foramen Monroi continue to grow fairly normally and that the parts lying proximally are either inhibited so severely as to enable one to speak of a defect (infundibulum region) or less severely (such as the cortex), it follows that the forebrain vesicle, which has continued partly membranous, remains lying halfway under parts which are growing normally. Infundibulum remnants and opticus remnants fix this vesicle to the base of the brain, which is also severely inhibited and defective. The result is that the thalamus in growing overlaps it and pierces itself, as it were, in a forward direction. Davidson Black's case can also be explained in this way without great difficulty. Winkler's case shows very little ventral displacement; it was, however, a much less severe case than ours. Indeed, it is striking that according as ophthalmic and facial deviations increase in severity, the displacements increase. We imagine that in a case such as that described by Winkler the connexion between diencephalon and telencephalon was only slightly injured (he also found a corpus striatum, which we did not find in any of our cases); there was no membranous degeneration of the margo thalamicus of His. Probably, therefore, there was here a less extensive change in the shape of the base of the skull and less membranous degeneration in the forebrain vesicle. The reason why the ventricle roof has become so large is a further question. In Davidson Black's case it is found that the aquæductus Sylvii has closed up. It is necessary also to postulate the influence of the pathogenic agent upon the material closing the tube. One should think of compensatory ependymal increases, to which von Monakow often drew attention, and finally of the possibility of a vacuum which has arisen.

In our opinion a teratologist cannot say much more about these matters. He cannot solve the question: defect or inhibition. He raises again and again the question as to the anatomical conditions in the front wall of the neural plate, but finds no solution for it in these pathological products and he is likewise unable on the ground of these few data to pronounce an opinion on the point whether such connexion applies generally. It is likely that diverse kinds will be met with among cyclopes as well as among anencephalics.

THE PINEAL BODY

The epiphysis of the synotus requires special discussion. The peculiaritics which one finds in this case suggested to Prof. Winkler the need that it should be more closely examined. In doing so he pointed to the analogy with the epiphyseal organ of reptiles. The diencephalon is distinguished from the other parts of the brain by the lobes which it forms and by which the brain is in communication in various ways with the outside world: the eyes, infundibulum, paraphysis, glandula pinealis and parapineal organ. Occipitally the roof plate forms two little organs which are mostly distinguished. The hind lobe is the analogue of the epiphysis (glandula pinealis):

in the case of petromyzon and anura a kind of eye has been formed out of the top of the vesicle and lies immediately under the skin, which has become transparent. This little organ is connected by a nervus pinealis with commissura posterior and right ganglion habenulæ. In higher classes of animals the lobe is less elongated, the walls collapse and the gland tissue of the glandula pinealis develops. Pedes conarii are the analogues of nervi pineales. Immediately in front of the epiphysis there is found in petromyzon and lacertilia a second lobe which conducts itself correspondingly, with formation of a ' bladder ' and a nerve which brings about a connexion with the other ganglion habenulæ. This eye was probably of great significance in extinct reptiles. After all, it is not precluded that both organs are of equal significance and form a pair of dorsally directed eyes, one of which does not develop any further.

In our case little of the gland tissue of the glandula pinealis is found. In its stead the hind part of the third ventricle is covered here with a palisade epithelium, underneath which fibres having myelin taper off on all sides. These form pedes conarii which turn ventrolaterally towards the place where the corpus quadrigeminum anterior should lie. Which organ is this ? The supposition that it is the glandula parapinealis (i.e. the parietal eye) seems at first sight to be far-fetched : where then has the glandula pinealis itself remained, one might ask. Is it a much altered epiphysis ? This too, cannot be said with certainty. Certain comparative anatomists do not, for that matter, see in petromyzon any sharp contrast between pineal and parapineal organ ; both are closely adjacent lobes of the third ventricle roof, both send fibres to the ganglia habenulæ, both penetrate frontalwards as far as the skin. If this standpoint is adopted, no useful purpose would be served by inquiring which of the two pineal eyes has made its appearance here, because both have the same significance. At any rate we find here a form which never occurs either in young or in fullgrown mammals. It is in point of fact a form which is seen like this only in lower animals such as reptiles and petromyzon. In the second place it is a remarkable fact that the fibres of this organ not only go to the ganglion habenulæ, but also disappear in the corpus quadrigeminum. If we recall to mind the anatomy of the diencephalon in its entirety, we see here the absence of everything that relates to the eye, a renewed appearance of an eye-like organ which sends fibres to the ganglia of the opticus system. This is indeed a remarkable change, a striking instance of the effect of syneidesis in the formative instinct (von Monakow). In this attempt at restoration older forms therefore make their appearance phylogenetically, and the restoration takes place at a lower level.

SIZE AND SITUATION OF THE NUCLEUS RUBER

We examined more carefully the size of the nucleus ruber in the case of cyclopia and compared it with a normal red nucleus of a new-born child. On

cursorily looking at the series, we received the impression that this nucleus was unusually large (like the oliva inferior). Measurements of the surface, as exact as was possible, confirmed this impression ; in some places the nucleus is larger than the normal is anywhere. However, if account is taken of the axial extension, this enlargement proves to be merely local. In the diagram (fig. 24) the surfaces at various levels can be compared with each other. Thus we find that in the cyclops the red nucleus extends to a shorter distance from the mesencephalon (absolutely as well as relatively, compared with a normal nucleus ruber). We find that the nucleus ruber in its entirety is smaller than normal ; this is due to the short distance to which it extends notwithstanding its relatively great thickness. The displacement in the direction of the cerebellum points to the possibility that the nucleus, which receives no impulses from the frontal parts of the brain and from the brainstem, has been displaced towards the normal cerebellum. It is also possible that in the

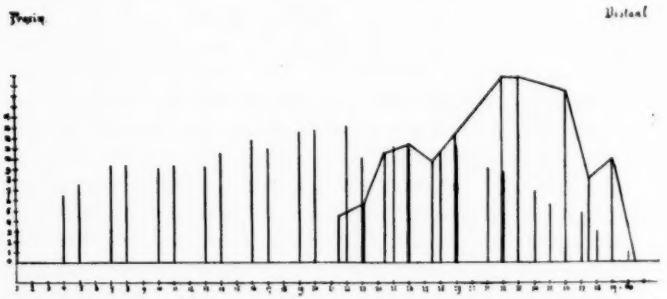

FIG. 24.—Diagram of surfaces of a normal nucleus ruber (neonatus) and the cyclopian nucleus ruber.

cyclops the mesencephalon has remained too small in its entirety. The displacement of the red nucleus is, indeed, a consequence of the axial shortening of the mesencephalon. We are inclined to accept the latter explanation because it accounts sufficiently for the deviations which have been found and because the importance of neurobiotaxis to teratology has up to the present been found to be very doubtful. (See Winkler's criticism in his *Handbook*, Vol. III, p. 9, and Schenk, *Z. Neur.* 146.) It is extremely difficult to form an idea of the displacement of a nuclear 'Anlage' before there can have been any question of stimulating influences of centres : these have not yet developed in the 'Anlage.' However, we more frequently find compensation symptoms than adaptation symptoms and it is accordingly conceivable that, in the absence of all higher centres, parts of the nucleus ruber, which here is one of the most proximally situated reflex centres, have developed strongly in contrast with its environment, which already shows signs of injury. This applies still more to the oliva inferior.

In the medulla oblongata of normal size, enlarged olivæ inferiores have

developed (fig. 25). The cerebellum, too, seemed much enlarged. With the aid of calculations of the surface it was possible to estimate the nucleus ruber as well as the oliva inferior in their entirety. The calculated contents of the oliva inferior was found to be fully 157 c.mm. in the normal neonatus, and fully 383 c.mm. in the cyclops (i.e. a proportion of 10 to 24·4).

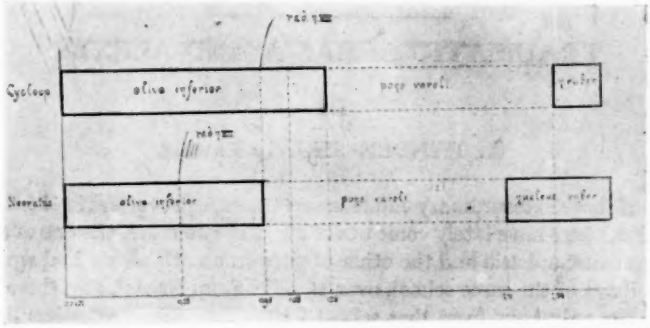

FIG. 25.—Diagram of the axial distension of oliva inferior, pons and nucleus ruber in human cyclopia and a normal neonatus.

The contents of the region of the nucleus ruber amounted in the normal neonatus to slightly less than 42 c.mm., in the cyclops to 9 c.mm. (i.e. a proportion of 10 to 2·14).

Owing to the inaccuracies which result from this method of calculating, these figures are not reliable in every respect. However, the comparison of these values, which have all been obtained in the same manner, is of great importance and reveals gross deviations.

Short Notes and Clinical Cases

TRAUMATIC APHASIA AND ALEXIA

By

A. RENDLE SHORT, Bristol

By one of those extraordinary coincidences that are proverbial in the medical profession, there have lately come under my care two cases, the one of almost pure traumatic aphasia and the other of pure traumatic alexia and agraphia, in schoolboys of the same school, over 40 miles from Bristol, and these are, I believe, the only boys from that school I have ever seen professionally. In both cases the disability was only partial. Both were due to a kick at football. In neither was there an open wound; in both there was a localized fracture of the skull. One was operated on for depressed fracture; in the other case the fracture was not depressed and no operation was performed. Both made a good recovery.

TRAUMATIC APHASIA

Case 1.—Patient, a boy of 16, was seen on November 16, 1932, in consultation with the late Dr. Cameron of Taunton. Four days before he had been kicked on the left temple during a football match. He was unconscious for 20 minutes. As soon as he recovered, it was noticed that he had considerable difficulty in talking, though he was able to understand everything that was said to him, and to read to himself. There was some weakness of the right side of the face of supranuclear type. Headache passed off after a day or two.

When seen at a nursing home, the boy looked bright and had no headache. Pulse and temperature were normal. There was no facial paralysis now on smiling or shutting his eyes, but it was still visible when he spoke. There was tenderness over the left temple, but no depression of the bone could be felt. There was no paralysis, squint or nystagmus.

Patient remembered past events well and could reply to questions better than on the preceding days, making no mistakes. The aphasia had largely cleared up. Asked, however, to repeat 'To be or not to be,' from Shakespeare, which he said he knew well, he stumbled over the words a good deal. He could read.

The skiagram showed a small depressed fracture over the site of Broca's convolution. The left side of the skull was exposed by operation over the depressed area, and a depression of both tables, about the size of a shilling, found. The depressed bone was elevated and removed. There was a little extradural clot beneath it, which was removed. The dura mater pulsated well; it was not opened.

Patient made an uneventful recovery. In a few days the aphasia had gone. He was seen in 1935, well in all respects.

TRAUMATIC ALEXIA

Case 2.—J.T., age 14. Seen on February 17, 1936, with Dr. Pearse of Taunton. Two days previously, whilst playing Rugby football, he was kicked on the left side of the head. He appeared to be dazed for a few seconds, and the space of about 15 minutes was missing from his memory. He was put to bed and vomited several times, but there was no headache at any time. There was bleeding from the left ear for 24 hours. That morning, a letter arrived from his mother and he was unable to read it.

On examination, patient was a normally developed boy of average intelligence, quite bright and cheerful. He complained of no pain, and said that his sight was normal, except for the difficulty in reading. He talked normally and understood everything that was said to him. There was a tender area, slightly swollen from bruising, the centre of which was $2\frac{1}{2}$ inches above and $1\frac{1}{2}$ inches behind the left auditory meatus. A skiagram showed a small stellate fracture at the site of tenderness, without depression of the skull. Temperature had varied from 97·5° to 98° and pulse from 60 to 76. There was no muscular weakness, no loss of sensation, no abnormality of reflexes; eyes were normal, optic discs normal. He counted figures at once and accurately, and recognized objects at sight. He recognized a large number of pictures from a book correctly, except that a cap he called, 'what a boy wears on his head.' Asked to read the number-plate on a picture of a motor car he read the four figures at once, but called the letters S.D. instead of R.D. He read simple printed script stumblingly, missing about one word in four, e.g. he could not read 'lorry.' He read the French verb je vais (pres. indicative), which he knows, correctly, but read 'nous' for 'vous' in a sentence. He could not read Latin. He wrote the headmaster's name correctly, but misspelt his own. He wrote his age 14, but could get no further than 'fourtee' in letters. He wrote from dictation, 'The Lord is my Shepherd' (his spelling and reading are said to be normal for his age). He appeared to be green-blind, but recognized other colours well, even in dots no bigger than printed letters.

The headmaster undertook to test his reading day by day and report to me.

On February 18 he was given Mark i. 1–5. The following words were either misread or unread: Gospel, behold, messenger, which shall prepare thy way, voice, crying, wilderness, make his paths straight, baptize, preach, baptism, repentance, remission, there went out, him, Judaea, they, Jerusalem, baptized of him, confessing their.

On February 19 he was tried with a vertical list of three-letter words for 15 seconds. He read ten words with four mistakes, reading two w's for x's. He could only read four out of a vertical list of four-letter monosyllabic words in 15 seconds.

On February 20 he could read boys' names—Tom, Jack, Archibald, at once; Gerald had to be written for him.

On February 22 he read a vertical list of three-letter words with three mistakes out of seven, and five four-letter words, all incorrectly. He was, however, able to write them and then read them.

On February 29 and March 1 he read Mark ii. 22 to iii. 24 correctly. It is evident, therefore, that the alexia has now cleared up.

COMMENT

Localized injury of the cerebral cortex, as a result of a blow on the head, is not uncommon in surgical practice, with or without a depressed fracture of the skull, but usually the symptoms are those of a lesion of the motor or visuosensory areas. One patient under my care, a public schoolboy, had a

transient hemianæsthesia of the right side of the body after a blow over the left parietal bone; there was no period of unconsciousness, and the trouble cleared up in a few hours. But almost pure aphasia or alexia following such an injury must be quite rare, and appears to be of considerable physiological interest. The centre of the blow in the first case was over Broca's convolution, and in the second case over the area generally supposed to be connected with the function of reading.

The question of prognosis, and, depending on that, of treatment, is interesting. So much improvement had already taken place in the first case that it seemed all but certain that recovery would be perfect whether operation was performed or no. As there was definite depression, however, we thought it better to elevate the bone so that it might not be a focus of irritation later. In the second case there was no bony depression, and up to the time of examination no improvement had been observed; but, encouraged by the fact that the alexia was only partial, that general experience shows that in non-fatal local injuries of the brain a certain amount of recovery is to be expected, and remembering the happy course of the cases of the two other patients mentioned, we decided to adopt expectant treatment. As will be observed, after a fortnight from the date of the accident the boy could read correctly.

Editorial

AVITAMINOSIS AND THE NERVOUS SYSTEM

IT is almost forty years since Eijkman, the medical officer of a Javanese prison where beriberi was rampant, observed that fowls feeding on refuse rice developed paralytic symptoms like those of his patients, and showed that pigeons fed on polished or decorticated rice were attacked whereas others eating the whole grain continued immune. Some essential food factor, thought by him to be protein and phosphorus, evidently disappeared when rice was submitted to a shelling process. The identity of this 'polyneuritis gallinarum' with human beriberi was assumed. In 1907 Holst proved that the deficiency could not be one of protein by quoting an outbreak among Norwegian sailors enjoying a diet rich in meat. To Funk (1911) is due credit for the preparation from rice polishings of a crystalline substance having a pronounced curative effect on avian polyneuritis. From these days the lore of avitaminosis has steadily been incremented; the word 'vitamin' has become popular as a term for a mysterious substance that has a miraculous effect; and the profession, captivated by the same notion, is now seeking an explanation for a variety of nervous symptoms and diseases in the negative factor of absence of something. That this way scientific danger lies can hardly be gainsaid.

So long ago as 1912, when at a medical society meeting Funk claimed, and indeed showed, that the paralysis of pigeons fed on hulled rice could be made to disappear in an hour or two by the simple addition of whole rice, the criticism was advanced that whatever this paralysis was, it could not possibly be due to a polyneuritis in the sense of the neurologist. It must rather have been a dynamic defect or cessation of function due perhaps to some kind of cell starvation. Since that date, a number of studies have culminated in the view that so far as this alleged polyneuritis of pigeons is concerned only the acute symptoms can be made to disappear dramatically by injection of extract of rice polishings, and these acute symptoms are not those of any kind of neuritis. On the contrary, they are rather of the

nature of tonic spasms and so-called vestibular fits, for which an origin in mesencephalon or brainstem—as in a decerebrate animal—is much the most likely. Other studies have finally led to the isolation of the substance prepared by Funk, which is now named the ' antineuritic ' factor vitamin B_1. According to Peters its formula (or rather that of its hydrochloride) is $(C_{12}H_{17}ON_4S \cdot HCl)Cl$. A daily dose of 0·002 mgm. (2·0 γ)—constituting the international vitamin B_1 unit—will cure pigeons suffering from experimental ' polyneuritis.' Peters and his colleagues have shown, by a series of fine researches, that the rôle of the vitamin concerned is to help in oxidizing certain carbohydrate intermediates (notably lactic and pyruvic acids) found principally in the brainstem ; it is a catalyst whose absence will interfere with the metabolism of glucose, failure in the development of energy from sugars stopping the normal activity of various central groups of nerve-cells. If this is so, if the ' biochemical lesions ' of vitamin B_1 deficiency are in reality pontobulbar, then the inference would be that the acute and reversible symptoms of avian ' polyneuritis ' are in reality of central origin ; a supply of B_1 will remove pyruvates and quickly restore tissue respiration. But it must be equally clear that the experimental data do not account for the fact that the symptoms and lesions of beriberi are in essence neuritic ; why and in what way impeded carbohydrate metabolism should affect peripheral nerves, as contrasted with nerve-cells of the brainstem, does not appear, and has not been explained. Through all Peters' experiments the action of the morbid process is seen to be on nerve-cells, whereas in human beriberi these are scarcely changed, the maximum effect falling on distal peripheral nerves. Again, various efforts to reproduce ' neuritis ' by feeding rats and other animals on a dietary lacking the ' antineuritic ' substance B_1 have been extraordinarily inconclusive. In spite of these and other reasons for suspending judgment, we note an increasing tendency nowadays to ascribe other forms of neuritis not to a specific (or unspecific) poison but to inadequate supply of vitamin B complex ; this has been asserted in regard to the alcoholic variety, that of pregnancy, and others still. Some writers go to the absurd extreme of alleging that these kinds of neuritis, and beriberi, are one and the same disease, ignoring the self-evident difficulties of such a claim—for instance, the geographical factor is overlooked. As Aykroyd asks, why should a wretched diet of milled cereals lead to pellagra in South Carolina, to beriberi in Java and Newfoundland, and to scurvy

on the Rand ? And we may ask, too, on general lines, what is the precise causal connexion between vitamin and disease ? Does the latter spring into being as soon as ever the former fails, without the action of any intervening process ? Were this so, the observed fact that only a proportion of those persons whose food is insufficient develop symptoms becomes incomprehensible ; in an Egyptian native family, for example, there may be, indeed very frequently is, one solitary pellagrin, yet all the other members are eating the same food, drinking the same drink, living in the same surroundings, having the same agricultural occupation. In the case of rickets there is good reason to believe that the relation between vitamin and disease is far from simple, and data advanced above show this to be true (mutatis mutandis) of beriberi.

Another rather curious circumstance is the constancy with which ' cures ' are vaunted for proof of the hypothesis that lack of a foodstuff per se is responsible for all the phenomena, clinical and pathological. This claim is always being put forward, e.g. by those who think that the neuritis of pregnancy or of alcohol is due to shortage of the ' antineuritic ' factor. There is no notion here of the possible function of vitamin as a shielding or defensive substance, in the absence of which some noxa runs riot. Yet cogent evidence might be adduced to suggest that the purpose of vitamin in the organism is prophylactic. The idea that ' naturam morborum curationes ostendunt ' is an old fallacy, and it is odd to find it cropping up once more in connexion with avitaminosis. Were it correct, then the fact that hysterical syndromes can be removed (say) by priestcraft and by psychoanalysis alike must signify that the psychoneurosis is due to demoniacal possession as well as to an abnormal vita sexualis.

The position at present in respect of avitaminosis seems to be, that the complexity of the pathogenic factors is being ignored in some quarters, and that a false simplicity has been introduced by use of such unfortunate terms as ' antirachitic ', ' antineuritic ', ' antixerophthalmic ' and other ' anti ' vitamins, as though there were specific vitamins for specific diseases. The number of the former is doubtless slowly increasing, but their rôle in pathogenesis has yet to emerge in complete and definite form.

Abstracts
Neurology

SENSORIMOTOR NEUROLOGY

[1] **The epilepsies.**—FOSTER KENNEDY. *Amer. Jour. Psychiat.*, 1935, **92**, 527.

THE phenomena of epilepsy are apparently responses on the part of neural mechanisms to various forms of noxious stimulation. The kind produced depends more on the kind of function of the neural mechanism which responds than on the nature of the stimulus that produces the response. It is hardly much of an exaggeration to say that the normal individual of 50 cannot have arrived at that age without some form of epileptic phenomena having occurred in his life. This is a conception which might be set against that held by a great many doctors that epilepsy is necessarily a progressive, practically incurable, and always deteriorating disease. One might speak of the epilepsies as though they were a spectrum—a spectrum the colours of which blend the one into the other. In that spectrum is the great fit, following an ordered pattern of sudden unconsciousness and decerebrate rigidity, followed, apparently due to mesencephalic block, by the disordered resumption of control by the cortex, followed by exhaustion, followed by resuscitation. In that same spectrum are many other forms of seizure. By giving them special names we have obscured our own consciousness that eclampsia is epilepsy, that uræmia is epilepsy; and we find the epileptic effects of insulin changing the content of blood sugar as alterations in the metabolic rate of the brain. It is almost as if the brain were in a state of awareness against anything that would interfere with the proper oxygenation of the cell. Any form of deoxygenation of the cell increases the irritability of the cell and the energy discharge is a response to anoxæmia. This is true as seen clinically in the epilepsy of the alcoholic brain. It is true of the brain of the paretic. It is true of any increased water content of the brain, that the film of fluid interferes with the oxygenation of the cell and permits or precipitates the discharge of its energy. It is possibly true in another domain—an urgent response by neural centres to irritants producing localized brain œdema. Localized collections of fluid can produce such states of deoxygenation of cells as to produce an epileptic discharge. Undoubtedly, too, the vasovagal attacks of Gowers are discharges along sympathetic paths. We have, as it were, rediscovered the epilepsies of the sympathetic system. Further, the psychic equivalents are undoubtedly an expression of disordered integration of the brain—appearing as sensory or motor phenomena. The psychic

phenomena of epilepsy should suggest the somatic origin of many of the psychoses.

C. S. R.

[2] **Cortical autonomic epilepsy.**—J. W. Watts and C. H. Frazier. *Jour. Nerv. Ment. Dis.*, 1936, **81**, 168.

Two cases are reported in which autonomic manifestations (nausea, epigastric distress and vomiting) were often substituted for epileptic convulsions. From a study of these cases it would seem that both sensory and motor autonomic representation exists in the cerebral cortex of man, but exact localization is impossible in the present state of knowledge. Epigastric auras and other visceral sensations described by patients are not so-called referred pains, but the result of vigorous and perhaps abnormal movements of the gastrointestinal tract.

R. G. G.

[3] **A rare epileptic manifestation** (Una rara manifestazione di natura epilettica).—P. Serra. *Riv. di pat. nerv. e ment.*, 1936, **44**, 666.

The author describes as an epileptic equivalent a rare symptom which appeared in an epileptic subject. This was a recurring pavor nocturnus with a consequent motor reaction of unconscious flight. This was repeated during sleep whether by day or night and was intimately related to the fundamental epileptic state.

The author discusses emotional reactions during sleep in epileptics and their relationship to the actual epileptic attack.

R. G. G.

[4] **Catalepsy or flexibilitas cerea in a three-year-old child.**—A. Blau and S. H. Averbuck. *Amer. Jour. Psychiat.*, 1936, **92**, 967.

The case of a mentally retarded three-year-old child who showed the unusual symptom of flexibilitas cerea is here presented. Following a period of observation, the catalepsy gradually disappeared simultaneously with a progressive improvement in the intellectual status. It is suggested that this type of catalepsy is physiological or developmental in nature. The relationship to the general question of catalepsy is discussed.

C. S. R.

[5] **Little's disease with idiocy produced by the status verrucosus deformis** (Sindrome di Little con idiozia sostenuta da Status verrucosus deformis). *Riv. di pat. nerv. e ment.*, 1936, **44**, 628.

Little's disease, i.e. congenital spastic quadriplegia, shows at autopsy sometimes signs of cerebral alteration, sometimes spinal (agenesis of the

pyramidal tract); sometimes both parts are affected. In cases of primary spinal lesions the patients usually retain a normal intelligence, have normal facies and do not show squint or epilepsy. In other cases afflicted by more or less serious defect the lesions are generally found in the brain. In the present case a status verrucosus deformis in the brain was discovered. Hypoplasia of the medulla existed but was not sufficient to account for the gravity of the symptoms by itself; they seemed to be secondary to the destruction of cortical pyramidal cells. The case is similar to that described by Pellizzi, that of a patient with generalized spasm, epileptic convulsions, and idiocy, who was born at the seventh month. At post-mortem examination the brain showed microgyria in the neighbouring convolutions and arrested development of the pyramidal tracts.

With regard to the pathogenesis of the verrucosus condition Ranke has demonstrated that towards the fourth and fifth month of fœtal development before the appearance of the convolutions, an atrophic condition of the cortex might develop in special circumstances, assume a pathological appearance, and spread more or less on the surface, but also extend from the surface into the interior of the cortex. Ranke believes that such deformity is not always dependent on defects of development, but can be provoked by morbid processes. Kraepelin states that such cases are as a rule idiots with epilepsy. In the present case besides the idiocy and rare epileptic attacks there also existed a congenital quadriplegia. Bourneville stated that such lesions resemble those of tuberose sclerosis or fœtal encephalitis. It is probable that here the condition was provoked by a fœtal meningoencephalitis at the fourth or fifth month which began in the cortical zone and from there extended more or less to the whole brain. In this way may be explained the adherence of the meninges, the thickening of the vessels, the accumulation of neuroglia, the disordered cortical architecture and the presence of pathological cells. The ætiology of such fœtal meningoencephalitis is unknown.

R. G. G.

[6] **Neurological findings in mastoiditis.**—J. M. Nielsen and C. B. Courville. *Jour. Nerv. Ment. Dis.*, 1935, **80**, 541.

The writers studied 106 consecutive cases of clinical uncomplicated mastoiditis without intracranial extension and found that already many neurological signs were present. They discovered alteration in normal reflexes, mild eyeground changes, pathological reflexes, focal headaches, pupillary changes, pain in the eye of the same side, vomiting or severe nausea, nystagmus and meningeal signs in that order of frequency. They do not find in their series of cases any aphasia, cerebellar symptoms, extraocular palsies, perimetric changes, focal convulsions or pareses.

R. G. G.

PROGNOSIS AND TREATMENT

[7] **Summary of the Report of the American Neurological Association Committee for the investigation of sterilization.**—ABRAHAM MYERSON. *Amer. Jour. Psychiat.*, 1935, **92**, 615.

KNOWLEDGE of human genetics has not the precision or amplitude which would warrant the sterilization of people *who themselves are normal* in order to prevent the appearance, in their descendants, of manic-depressive psychosis, dementia præcox, feeblemindedness, epilepsy, criminal conduct. An exception may exist in the case of normal parents of one or more children suffering from certain familial diseases, such as Tay-Sachs amaurotic idiocy. There is at present no sound scientific basis for sterilization on account of immorality or character defect. Until and unless heredity can be shown to have an overwhelming importance in the causation of dangerous antisocial behaviour, sterilization merely on the basis of conduct must continue to be regarded as a 'cruel and unusual punishment.' Nothing in the acceptance of heredity as a factor in the genesis of any condition considered by this Report excludes the environmental agencies of life as equally potent and, in many instances, as even more effective. Sterilization can only be recommended in selected cases of certain diseases and with the consent of the patient or those responsible for him. It may be considered in Huntington's chorea, feeblemindedness of familial type, schizophrenia, manic-depressive psychosis, and epilepsy. It is to be stressed that no great or radical change in the complexion of society can thus be expected.

C. S. R.

[8] **Results obtained by injections of 'betaxin' (vitamin B_1) in neurological cases** (Erfahrungen mit Betaxin (Vitamin B_1-Præparat) in der Neurologie).—H. NEUMANN. *Münch. med. Woch.*, 1935, **82**, 1959.

SUBCUTANEOUS injection, as well as intramuscular or intravenous injections, of betaxin 1 (Bayer) gave good results in cases of polyneuritis. Betaxin 1 is a crystallized vitamin B_1. Improvement was also obtained by injections of betaxin 1 in various diseases of the central nervous system—e.g. tabes dorsalis, subacute combined degeneration, amyotrophic lateral sclerosis and disseminated sclerosis.

M.

Psychopathology

PSYCHOLOGY

[9] **The propensity of genius to solitude.**—WARREN C. MIDDLETON. *Jour. Abnorm. Soc. Psychol.*, 1936, **30**, 325.

AN attempt has been made to show that some of the traditional arguments that have been advanced to prove the solitariness of genius per se are not

strongly convincing. A study of case-histories would not appear to lend support to any dogmatic pronouncement that men of genius are characterized by solitary traits, although it does seem justifiable to conclude that in some (perhaps many) geniuses there is a decided propensity to solitude and seclusion. This, however, does not seem to involve an innate disposition; rather, it would appear that the tendency can be accounted for on the basis of conditioning. Solitude is not the goal of genius but its refuge. Aloofness is frequently the result of a shrinking from the deteriorating standards of society. The genius is not only constantly forced into solitary retreat, but he sometimes seeks seclusion voluntarily and deliberately because as he associates with mankind he senses the advantages to be gained from insulating himself from the masses. In some instances contempt for society among men of genius may be the result of an attempt to compensate for a feeling of social inferiority.

C. S. R.

PSYCHOSES

[10] **Traumatic psychoses.**—Clarence A. Bonner and Lois E. Taylor. *Amer. Jour. Psychiat.*, 1936, **92**, 763.

Traumatic psychoses are rare, as in the Danvers State Hospital over a period of 16 years they numbered only one-half of 1 per cent. of first admissions. The factor of overindulgence in alcohol had a definite relationship both to the causation of the accident and to the adjustment of such patients after leaving the hospital. The type of injury sustained seemed to play an important part in the adjustment to former surroundings. The cases of fracture of the skull were less apt to readjust socially than those without fracture and this should be an important consideration in prognosis. The most common of the sequelæ appeared to be: continued irritability; headache; giddiness; and, to a lesser extent, irresponsibility, with impaired judgement. In the deteriorative forms, there was found immaturity and disturbances of the intellectual faculties. It would seem that only a small proportion of head-injury cases develop psychoses. It is well known, however, that they do not escape entirely from mental or nervous defects and that a large number develop neuroses, which ordinarily remain as such and do not develop into psychoses.

C. S. R.

[11] **Auditory hallucinations in prison psychoses.**—Rudolph Schwarz *Psychiatric Quarterly*, 1936, **10**, 149.

One hundred cases of prison psychosis were investigated as to the content of the hallucinations. Such hallucinations are symptomatic of a state of fear,

anxiety, a feeling of insecurity and apprehension. The peculiar content in prison psychoses seems to suggest that hallucinations are the outcome of a previous psychological experience. The feeling tone in such a psychotic individual seems to be the conditioning factor in regard to auditory hallucinations.

C. S. R.

[12] **The agglutinations of B. coli by the serum of psychotics, especially of schizophrenics.**—A. Beck, W. Ogden, and M. Whelen. *Jour. of Ment. Sci.*, 1935, **81**, 524.

The B. coli agglutination test with 76 sera of psychotics did not show, either in the frequency or in the intensity of positive reactions, any different behaviour from that observed in normal persons. The occurrence of normal agglutinins in schizophrenics was found to be nearly twice as frequent as in other psychotics. The observations did not make probable a causative connexion between B. coli infections and mental disease.

C. S. R.

PSYCHOPATHOLOGY

[13] **Purposive accidents as an expression of self-destructive tendencies.**—Karl A. Menninger. *Internat. Jour. of Psycho-Analysis*, 1936, **17**, 6.

The phenomenon of *apparent* (i.e. consciously) absent intention exists in acts which gratify deeper hidden purposes. In many of these accidents the damage is inflicted not upon someone else but upon one's own self. The body then suffers damage as a result of circumstances which appear to be entirely fortuitous but which in certain illuminating circumstances can be shown to fulfil so specifically the unconscious tendencies of the victim that we are compelled to believe either that they represent the 'capitalization' of some opportunity for self-destruction by the death instinct or else were in some obscure way brought about for this very purpose. The significant and differential thing about purposive accidents is that the ego refuses to accept the responsibility for the self-destruction. Illustrations are given of how a man plans a trap for another man to protect his home property, forgets that he has done so, returns after an interval, goes into the place he has so carefully protected and is himself killed or wounded. Psychoanalytic studies indicate that the unknown marauder represents a particular person in the unconscious phantasies of the murderer-victim. Automobile accidents often occur in circumstances which are suspiciously indicative of at least unconscious intent. In those cases where serious accidents take place because the driver has fallen asleep, they are probably sometimes determined by unconscious

suicidal impulses. There is also a similarity between the fatal accidents and those in which only a part of the body is destroyed. We may be able to see precisely how the accident serves to punish the individual for guilty acts or wishes. In those cases which are not fatal, however, this punishment serves not only as the price of atonement but as a permission for further indulgences in the same guilty acts or phantasies. The guilty act stimulates the conscience to demand of the ego a price. In some instances this price is a (self-inflicted) death penalty. In other instances, however, it seems to be less severe. One may note the principle of periodic payment for the continued indulgence in forbiddden erotic or aggressive tendencies in many neurotic patients, and melancholia is often forestalled or deferred by various obsessive and compulsive techniques. There are certain individuals who seem to fall victim to successive disasters with an uncanny regularity.

C. S. R.

[14] **Pituitary disturbances in behaviour problems.**—Matthew Molitch and S. Poliakoff. *Amer. Jour. Orthopsychiat.*, 1936, **6**, 125.

Ninety-seven boys with pituitary dysfunction in a State Home were studied and are reported from the standpoint of diagnosis, mental level, school achievement, behaviour, personality, and treatment. Those with such dysfunction were found to be brighter than the control group but the school attendance and achievement were the same in both. They were found, too, to be unstable, immature and suggestible. More personality deviations were found than in the controls. Within the Institution the 'pituitary' boys were found to be above average in school progress but below in conduct and adjustment. The offences causing commitment to the Home consisted essentially of stealing and truancy. Positive correlations were found between height and mental level. Five boys with hypopituitary dysfunction were treated with growth hormone with subsequent increase in height and improvement in secondary sexual development.

C. S. R.

[15] **Mental changes following head trauma in children.**—Abram Blau. *Arch. Neurol. and Psychiat.*, 1936, **35**, 723.

The cases of 22 children showing mental changes following head injury are reported. The conditions are classified and designated as posttraumatic acute psychosis, chronic behaviour disorder, epilepsy with secondary deterioration, and defect conditions and secondary intellectual deterioration. The acute psychosis was observed in six children. The onset occurred immediately after the patient recovered consciousness and the symptomatology consisted of a demonstration of unrestrained instinctual, emotional and

motor behaviour, associated with an affect of fear and anxiety. Complete recovery occurred in a few weeks. The chronic behaviour disorder was noted in 12 and closely resembled postencephalitic behaviour disorder. The syndrome is characterized by hyperkinetic, uninhibited, asocial behaviour, with an instinctual colouring. Of the nine cases in which X-ray examination of the skull was made, there were five in which fracture occurred in the frontal region and four in which no evidence of fracture was present. Delinquency was present in all cases. The intelligence as measured by the Stanford-Binet test was within the normal range. The general prognosis was poor and many required prolonged treatment in State Hospitals for patients with mental disease. Posttraumatic epilepsy occurred in five cases. The observations indicated that frequently it leads to behaviour disorders and to ultimate intellectual and emotional deterioration such as that which occurs in other forms of epilepsy. Mental deficiency is a rare sequela of head trauma, although a form of secondary intellectual deterioration may appear as a result of a lack of interest and attention.

C. S. R.

[16] **Sinus sepsis and mental disorder.**—R. E. JOWETT. *Jour. of Ment. Sci.*, 1936, **82**, 28.

A SERIES of 500 routine cases of mental disease were examined. Nasal sinus infection was proved in 7·6 per cent. This percentage compared with a similar one found in a similar series of cases using similar criteria shows only a slight difference. In a control series of 184 mentally normal individuals the incidence of sinus suppuration was found to be not less than 5 per cent. Pioneer workers in this field have claimed a higher incidence. It would appear from the above figures that the incidence of sinus in mental disorder is not greater to any appreciable degree than among the general population. That such sinus disease may seriously affect existing predispositions to disease in the nervous system, just as it may affect such predispositions in any other system, is unquestioned, in view of clinical and post-mortem work carried out.

C. S. R.

[17] **The forensic aspect of malingering** (Zur forensischen Bedeutung der Simulationsfrage).—F. KNIGGE. *Zeits. f. d. g. Neurol. und Psychiat.*, 1935, **152**, 163.

ALTHOUGH an exact psychological basis has not been traced for the diagnosis of malingering, sufficient cause may be determined in a particular case so that the medical expert will be able to give this diagnosis without scruple. Malingering by prisoners is often observed not as a result of mental disease,

but as a criminal act committed in order to prevent penalty. The presence of a psychopathic disposition does not preclude the diagnosis of malingering, as supposed by some observers. A ' sham ' patient has not to be cured, but in certain circumstances at least has to be punished. The transition from malingering to a state of morbid hysteria is prevented by intensive attention to such a person.

M.

[18] **Some recent investigations into the hæmatology of the psychoses.**—H. C. Beccle. *Jour. of Ment. Sci.*, 1935, **81**, 840.

From his work the writer concludes that the toxic and Sudanophil granule estimations appear to follow the course of the psychosis, varying directly with the intensity of the disorder. The gradual improvement in the counts observed in recovering cases must be ascribed in part to the general detoxication which is instituted in each patient soon after admission. Though it would be rash to infer from only a few examinations that these estimations have definite and prognostic value, it is suggested that they may prove of considerable assistance.

C. S. R.

PROGNOSIS AND TREATMENT

[19] **Hæmatoporphyrin treatment of severe depressions.**—D. Louis Steinberg. *Amer. Jour. Psychiat.*, 1936, **92**, 901.

Theories as to the physiological action of the drug are by no means clear. Laboratory studies had shown a tendency of the drug to elevate the blood count and to reduce the calcium level. Fourteen patients in all were treated. Two responded well, one of whom relapsed after a period of six weeks. The other continued to adjust on a low level. Laboratory examinations failed to disclose any specific influence on the blood picture except a tendency to lowered cholesterol levels. The results did not warrant the use of this therapy in preference to any of the old armamentarium used in the treatment of depressed psychoses. No deleterious toxic reactions were noted.

C. S. R.

[20] **The hæmatoporphyrin treatment of depressive psychoses.**—Leslie R. Angus. *Amer. Jour. Psychiat.*, 1936, **92**, 877.

No satisfactory explanation, based on experimental evidence, of the action of hæmatoporphyrin has yet been evolved. Laboratory tests are of little value in selecting cases for treatment or gauging their progress under treat-

ment. Blood calcium is reduced by the drug whether the case be clinically improved or not, but both before and after treatment is usually within normal limits. Blood sugar tolerance curves assume a more normal configuration after treatment than before it. Blood sugar falls during treatment, but is within normal limits before and after it. The blood picture is not characteristically affected. In only three cases was any continued fall in weight apparent. No toxic effects were observed and with one exception no clinically unfavourable sequelæ to treatment appeared. Improvement, if it does occur, takes place early. If one course fails, later series, even in much increased dosage, are not successful. In the author's cases clinical improvement established during a course of hæmatoporphyrin treatment appeared to be maintained after the drug was withdrawn. Taking everything into consideration, it would seem that hæmatoporphyrin is of definite value in the treatment of symptomatic depressions. While the results are unquestionably better in the milder cases, the method has proved of positive benefit in certain cases of apparently malignant character and would seem to be worthy of trial in any case in which depression is an outstanding symptom.

C. S. R.

[21] **The fate of 150 psychiatric out-patients.**—Ian Skottowe and Madeline R. Lockwood. *Jour. of Ment. Sci.*, 1935, **81**, 502.

Of 150 consecutive unselected psychiatric out-patients, only the psychotic and psychoneurotic patients (114) were suitable for active treatment. Of the 114, 28 refused to carry out what was advised and in 18 others the ultimate result could not be ascertained. In the remaining 68, 50 (70·4 per cent.) recovered or were much improved, 32 (47 per cent.) required mental hospital treatment and of these 24 (75 per cent.) were discharged recovered or much improved. Psychological treatment was applied in 13 cases and 11 (84·6 per cent.) were recovered or much improved. There were 23 who were dealt with at home under their own doctors, with attendance at the department for supervision and superficial psychotherapy. This group showed 17 (74 per cent.) recovered or much improved. The results of mental treatment were much better in those patients who received advice in the first instance at the psychiatric out-patient department than in the general run of mental hospital admissions. A striking point was the fact that schizophrenic patients formed the largest single group of those who refused to accept advice or to attend for treatment. Even allowing for this, the results in the less malignant forms of psychoses were better in out-patients than in the general run.

C. S. R.

[22] **A programme for the determination of the therapeutic effectiveness of the psychoanalytic method.**—Clarence O. Cheney and Carney Landis. *Amer. Jour. Psychiat.*, 1935, **91**, 1161.

It is proposed to select two groups of mental patients, each member of each group to be matched as nearly as possible with respect to age, sex, intellect, diagnosis, socio-economic status, etc., with a member of a second group. Both groups are to receive identical treatment, except that one group, the therapeutic, is to receive intensive psychoanalytic therapy. The other or control group is to receive little or no psychotherapy and no psychoanalytic therapy. Written statements of the condition of all patients at the beginning of the experiment, together with the probable prognosis, should be made. All patients should be followed for several years until the outcome of the therapy or lack of therapy is clear. From this study we should gain information concerning the diagnostic type, age, sex, intellectual status, etc., for which psychoanalysis is indicated or contraindicated. The frequency of success and failure of the psychoanalytic therapy could be stated. A basis for the relative value of the use of psychoanalytic therapy and methods in mental hospitals could be made.

C. S. R.

[23] **Effect of vitamins A and D and mineral administration in dementia præcox.**—J. Notkin, Frances Krasnow, Viola Huddart, William J. Thompson, and Lloyd E. Watts. *Amer. Jour. Psychiat.*, 1936, **92**, 925.

Fifteen women and 17 men, dementia præcox patients, were treated with vitamins A and D and a mineral mixture for a period of several months. An equal number of control cases was observed for approximately the same length of time. Biochemical determinations were made at the beginning and end of observation. One male sufficiently improved to leave the hospital, a female showed only a temporary improvement. Two others, one in each group, improved slightly but not enough to warrant discharge. It is questionable whether the vitamins were the responsible factors for the improvement; the psychological element cannot be disregarded. The remaining treated patients failed to show any change in their mental state. No change was noted in any of the controls. There was a higher incidence of gain in weight among the treated women as compared with those untreated. In the male group, however, the percentage of gain in weight was higher among the untreated control patients. The routine blood counts showed no variation of importance in any of the groups. No definite changes in the biochemistry of total lipoids, total cholesterol, cholesterol esters, free cholesterol, lecithin, albumen, globulin and calcium of the serum and of the total cholesterol and

lecithin of the spinal fluid took place as a result of the administration of viosterol and mineral tablets.

C. S. R.

[24] **The value and application of hydrotherapy in a mental hospital.**—Neil D. Black. *Psychiatric Quarterly*, 1936, **10**, 34.

Toxic and stimulative hydrotherapy by means of the needle shower and Scotch douche, followed by ultraviolet treatment, gives the best result in psychoneuroses and depressions. Eliminative hydrotherapy by means of the electric cabinet, needle shower, Scotch douche, and dry pack are best suited to toxic states. Their principal value is in the treatment of alcoholics, with the relief of the toxæmia; tonic hydrotherapy is indicated. Acute cases require daily treatment; following this stage, however, treatment twice or thrice per week is usually all that is needed. Length of treatment has to be determined by the needs of the individual patient, but those receiving eliminative, tonic, and stimulative types usually show a maximum of benefit after two months. Sedative hydrotherapy in the form of the continuous bath is indicated for prolonged cases of excitement. The wet pack is best suited for acute excitement of short duration. No time limit can be estimated for the treatment of acute excitement by such means; this must be determined by the patient's condition and his reaction to the treatment.

C. S. R.

Reviews and Notices of Books

Medical Aspects of Crime. By W. Norwood East, M.D., F.R.C.P., H.M. Commissioner of Prisons, etc. With a Foreword by the Rt. Hon. Sir John Simon. London: J. and A. Churchill. 1936. Pp. 437. Price 18s.

No one is better qualified than Dr. Norwood East to discuss with sympathy and sense a variety of problems bearing on the treatment of legal offenders and the relation of their misdemeanour or crime to their particular personalities. Constantly occupied with a special branch of his profession, he feels that a time has come when accumulated facts and impressions gleaned by him over a long period of years may usefully be shared with others, the outcome being this very readable and often deeply engrossing book. Its constituent chapters are mostly reproduced and amplified from individual communications, lectures, and addresses delivered at one time or another before varying audiences. About one quarter of the work is devoted to questions of prison administration; elsewhere 1,000 cases of attempted suicide are analysed; inter alia, the bearing of mental deficiency, drug addiction, and other abnormal mental states on crime is discussed dispassionately and ably. One of the most interesting chapters deals with murder from the point of view of the psychiatrist. Analysing 235 cases of murder, Dr. Norwood East found that only 35 of the murderers were insane or mentally defective, i.e. about one-seventh; and he quietly explodes a number of popular fallacies, such as that if a homicide confesses, or surrenders himself to the police, he is ipso facto insane, or that multiplicity of wounds has the same significance. He is no friend of the view that no murderer can be considered normal, that all such crime indicates insane impulse, that he who commits it needs treatment and not adjudication. Brushing away psychological speculations sometimes introduced to exculpate the accused, he concludes in words worth citing: 'the very existence of civilized society is endangered if crime is uncontrolled, and although the law in this country in regard to criminal responsibility is illogical, justice is done, and the manner in which it is done compels the admiration of the world.'

The Carotid Sinus and the Cerebral Circulation. By E. Ask-Upmark, Lund, Sweden. Supplement VI of Acta Psychiatrica et Neurologica. Copenhagen: Levin and Munksgaard. 1935. Pp. 374.

THE work represented in this comprehensive study of the carotid sinus—anatomical, experimental, and clinical—was carried out at the Harvard

Medical School during 1932 and 1933. It is a monumental contribution to a subject the importance of which has not yet perhaps been fully appreciated.

In every one of 61 animals representing 27 different mammalian species the carotid body was found, always localized on the internal carotid when that vessel occurs, just at the bifurcation of the common carotid. Abundantly supplied by twigs from ninth, tenth, and occasionally twelfth cranial nerves, as well as from the sympathetic, the first of these has most significance in relation to the sinus. From it vasomotor reflexes can be elicited by stimulation, and alterations in the bloodflow through pial arteries can be seen (by the ' window ' technique). These changes usually run parallel to the blood pressure, and this fact seems to indicate a passive relationship between systemic circulation and cerebral vascular system. The function of the sinus seems to be to guarantee continuity of the bloodflow to the brain ; it may also play a part in the response of the cerebral vessels to stimulations of vagus and sympathetic.

Clinically, irritation of the sinus is sometimes associated with syncopal attacks, which may be a feature of tumours of the carotid body. On the other hand, there is considerable doubt whether the mechanism takes any part in the discharges of epilepsy and none that surgical intervention on the sinus in such cases is unwarranted. Notice is taken of the connexion between changes in blood pressure in cerebral tumour cases and the relation of the site of the lesions to such change ; the occurrence of lowered pressure apparently depends on disturbance of some system in hypophysis and hypothalamus ; raised pressure seems connected with bulbar ischæmia.

Études Neurologiques. By Georges Guillain. Sixth series. Paris : Masson et Cie. 1935. Pp. 433. Price 80 fr.

The sixth series of Professor Georges Guillain's *Études*, composed of some 36 papers, once more testifies to the indefatigable industry and fine neurological insight of their distinguished author. He has set himself a high standard, and his contributions will stand comparison with any of those of former Maîtres in the Parisian school. Whatever subject Professor Guillain takes up he illuminates. A fascinating little study describes the occurrence of pressure on the external popliteal by the patient's (a sculptor's model) sitting immobile for hours at some 20 séances with right leg folded over left at knee, the nerve being pinched between peroneus and tendon of biceps cruris. A similar case occurred in a man who served in a bootshop and who, when fitting shoes for his customers, overflexed his left leg at the knee as he crouched on his toes, the customer's leg and foot resting on his left thigh. Many articles are devoted to cerebral and spinal tumour cases ; but the topics range in catholic manner over all the nervous system. The volume will prove a welcome addition to every neurological library.

Dictionary of Psychology. Edited by Howard C. Warren, Princeton University. London: George Allen and Unwin. 1935. Pp. 372. Price 15s.

THE compilation of a dictionary of 'technical terms met with in psychological literature or which the psychologist may wish to use in his writings' is a tremendous task. A long list of expert collaborators (we count over 110 therein) supplies a hint of its laboriousness and at the same time must beget confidence in the general accuracy of the result. In an appendix are placed tables relating to colour-vision tests, experimental errors, logical fallacies, statistical formulæ, and a dozen more topics of divergent kinds. There is a synoptic topography of the human central nervous system, and a catalogue of human reflexes 'frequently treated in psychological literature.' Prefixes and suffixes 'commonly used in scientific terms' are tabulated, and glossaries of French and German terms furnished.

Designed of course as a work of reference, the student can dip into it and is not likely to be disappointed in his search for any word, definition, or term that he comes across in his reading and for which he seeks a meaning or explanation. Opening it at random, we note that the first definition of 'neurosis' is: 'a functional disorder of the nervous system for which no actual lesion is found,' and at this interpretation possibly none will cavil; but an addition in brackets goes on to say: 'Current psychiatric usage tends to confine the term to psychogenic disorders, and to employ psychosis for disorders in which some chemical or physical cause is discoverable.' This statement we consider misleading. It is pleasing to note that 'drive' is preferred to 'urge,' which is defined as a 'strong tendency to perform an act'; the definition of 'epileptiform' is not that given by Hughlings Jackson, and is calculated to confuse, inasmuch as an 'epileptiform attack' is alleged to be due to 'some specific cause'—the implication being that an 'epileptic attack' is not. It is curious that 'aichmophobia' is not cited among the 'most common types of phobia.'

Taken as a whole, the dictionary is likely to prove helpful to all who consult it.

Sacrifice to Attis: A study of sex and civilization. By William A. Brend, M.A., B.Sc., M.D. London: William Heinemann Ltd., 1936. Pp. 350. Price 10s. net.

IN the worship of the Phrygian god Attis, emasculation was a customary ritual by the priests and such sacrificial rites became widespread in the ancient world, demonstrating the early repudiation of sex through religious influences. The thesis of this book is that mankind remains now dominated by similar irrational fears, sacrificing posterity thereby in the form of a falling birth-rate. Economic pressure as a causative factor is denied, the procedure being rooted

in ' man's primitive terror of the civilization he has created.' A reproductive instinct is regarded as a fallacy, for had there been one the ' disastrous ' fall of birth-rate could not have come about.

At some length religious influences are discussed. Though Christianity always preached ascetic idealism, it is thought that this was a product rather than a cause of sex inhibition and the conception of sex as inherently sinful is regarded as a man-made doctrine. This fear of sex in later centuries is dealt with, the allegation being that the Church has ' endeavoured to make universal a code of sex morals based in the last analysis upon nothing but fear.'

Many chapters are devoted to the various psychological factors of sex in its developmental stages and its relations to the origin of guilt, the conceptions of the father and the teaching of religion. Finally, Dr. Brend points out constructively that if the birth-rate is to be raised and the British race to continue, drastic measures will be needed. In the main it will mean the revision of the whole attitude towards sex enlightenment and freedom in the place of superstition and fear. The rights and responsibilities of youth must be recognized, parents and teachers must be alive to rational and scientific psychic training, hindrances to the development of a healthy sex life should be removed, freer intercourse between the sexes allowed, and greater freedom of marriage and divorce brought about.

There is, as a matter of course, much that is revolutionary in these pages, but throughout they make stimulating reading. The subject matter is of widespread interest both from a sociological and pyschological point of view and the author is eloquent in his treatment of it. The reader will find his mental horizon widened through its study.

Mental Nursing (Simplified). By O. P. Napier Pearn, M.R.C.S., L.R.C.P., D.P.M. With 22 illustrations. London: Baillière, Tindall & Cox. 1936. Pp. 328. Price 5s.

THIS serviceable little book, which in a compact way embraces all the different subjects of study needed for mental nursing training, appears now in a second edition with many additions, alterations, and improvements. The general plan of the well-known *Handbook for Mental Nurses* is followed. The author draws attention to the fact that a somewhat unusual style has been adopted, in that nearly every separate sentence expresses a fact that the nurse should know. For the purpose of reviewing all that has to be learnt for her examinations, the nurse should find these pages of great utility.

Hints to Probationer Nurses in Mental Hospitals. By Richard Eager, O.B.E., M.D. With 11 illustrations. London: H. K. Lewis & Co. Ltd. 1936. Pp. 222. Price 8s. 6d. net.

WHAT was originally a small volume has in this third edition been considerably enlarged and now constitutes something much more than mere hints

to probationer nurses. Purposely, the subjects of anatomy, physiology, first aid, hygiene, and sick nursing are not treated, attention being in the main given to mental nursing, normal and morbid psychology, and the various mental disorders. These are all dealt with in such a way as to create interest and understanding in a youthful nurse—a by no means easy task. The knowledge here given her will go far in helping her to carry on her duties intelligently.

Of especial value—and possibly not found in any other mental nursing work—is the information given on the malarial treatment of general paralysis of the insane and the special nursing that such patients need. It is also highly commendable that what nurses should know concerning mental hospital admissions and discharges is briefly added in a final chapter. In an appendix a selective list of books is given for those who may desire to further their knowledge and gain greater insight into all that appertains to their calling.

Though the contents are not free from errors—e.g. 'sublimation' has been wrongly defined—Dr. Eager has succeeded in producing a useful little book which merits a wide circulation.